FEB 2 3 1932

HUMAN BIOLOGY

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a record of research

FEBRUARY 1932

Vol 4 No 1

PUBLISHED QUARTERLY BY
THE JOHNS HOPKINS PRESS, BALTIMORE, MARYLAND, U.S.A.

HUMAN BIOLOGY

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HUMAN BIOLOGY is published quarterly by The Johns Hopkins Press, Baltimore, Maryland, U. S. A. The advance subscription price is $5.00 a year; foreign postage 25 cents extra. Single current issues are $2.00 each; back numbers, $3.00 each; back volumes, $6.00; plus postage. Subscriptions for less than a year are charged at the single issue price.

Subscribers should notify the publishers of change in address at least two weeks before publication of the issue with which change is to take effect. No claim for non-receipt of an issue will be entertained unless made immediately upon receipt of the next succeeding issue.

Manuscripts and communications regarding editorial matters should be addressed to Editor, Human Biology, 1901 E. Madison St., Baltimore, Maryland, U. S. A.

THE JOHNS HOPKINS PRESS, *Publishers*
BALTIMORE, MARYLAND, U. S. A.

Entered as Second Class matter, February 18, 1929, at the Post Office at Baltimore, Md., under the Act of March 3, 1879.

Vol. IV No. 1

HUMAN BIOLOGY

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FEBRUARY, 1932

THE NEGRO INFANT*

BY HARRY BAKWIN, M. D.

New York

Aside from the basic importance of a knowledge of the facts of health for the Negro himself, and of the influence of the health of the Negro on that of the community in general, the presence in the United States of over twelve million Negroes affords an opportunity to study the part played by race in influencing disease susceptibility and course.

Disease and death are physiological changes resulting from the reaction of the individual to environmental traumata, both remote and recent. Back of the immediate pathological condition are the physiological and morphological changes resulting from the action of environmental influences on the make-up of the individual. It is only by studying these various elements separately that one may hope ultimately to understand the different disease susceptibilities of different individuals.

The part played by sex in the health of the infant has been discussed elsewhere (3). In recent papers studies of body build in infants have been reported (4) (5) (6).

It has been our purpose in this study to assemble data on the differences between Negro and white infants in health and disease and to attempt to determine to what extent these differences are dependent on the element of race.

In 1851, Cartwright of New Orleans reported the results of a study made by a committee of which he was the head, on the "Diseases and Physical Peculiarities of the Negro Race" (10). He pointed out that the skin is not the only racial difference, but that the "Negro's brain and

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nerves, the chyle and all the humora are tinctured with a shade of pervading darkness." The Negro tolerates the sun's rays better due to "an anatomical peculiarity in the inner canthus, contracting the field of vision and excluding the sun's rays. . . . There is a deficiency of the red blood in the pulmonary and arterial systems from a defective atmospherization or arterialization of the blood in the lungs, ... a deficiency of cerebral matter in the cranium and an excess of nervous matter distributed to the organs of sensation and assimilation." It is this last anatomical peculiarity which accounts for the sensuality of the Negro.

"Drapetomania or the disease causing the slaves to run away" and "Dysaesthesia Aethiopis, or Hebetude of Mind and Obtuse Sensibility of Body—a disease peculiar to Negroes—called by overseers Rascality" occurs only in the Negro race. This condition is to be treated by anointing the skin with oil and slapping in with a broad leather strap.

In 1888 Morison (61) described the racial peculiarities of skin disease in the Negro. He pointed out the relative rarity of acne, eczema, favus, furunculosis, lupus, psoriasis and dermatitis venenata in the Negro.

In 1896 Matas of New Orleans (59) presented the results of a comprehensive study of disease in the Negro. He based his remarks on careful observation and statistical analysis of disease in approximately 64,000 Negro and white patients admitted to the Charity Hospital over a 10-year period.

Hoffman (39) in 1913 reported the results of a statistical survey of disease at Johns Hopkins Hospital over a 20-year period. His paper contains much important matter on disease morbidity and mortality in the Negro.

Within recent years numerous observers have pointed out the relative infrequency of chorea, rheumatic fever, angina pectoris (13) (82), diabetes (50) (29), exophthalmic goiter (64), and various forms of skin diseases (25) (34) in the Negro. Rosser (70) has written on peculiarities of rectal pathology in the Negro, and Day (14) on the urological idiosyncrasies. Scheppegrell (73) has pointed out the lesser frequency of otitic disease in the Negro.

Love and Davenport (55) compared the incidence of disease in white and colored troops admitted to sick report in camps in the United States during the recent war. They found the incidence of tuberculosis, pneumonia, smallpox, chicken-pox and venereal diseases much higher among

Negro than among white troops. Hysteria, drug addiction, epilepsy, and hemorrhoids were also more common among the Negroes. They were, however, relatively immune to acute inflammations of the skin, both infectious and toxic (snake-bite), to diphtheria, scarlet fever, German measles, influenza and naso-pharyngeal infections. Eye defects were few and the liability to refractive errors about one-half that of the white soldiers.

In a recent paper, Dublin (18) reviewed the experience of the Metropolitan Life Insurance Co. with nearly 2,500,000 Negro policy holders. He found the Negro death rate higher than the white at all age periods under 45. The excess was less pronounced during infancy than during any age period excepting the 5-14 year old group for males. The higher Negro mortality rate was due mainly to the high death rate from tuberculosis, organic heart disease, chronic nephritis, and pneumonia, with syphilis as a prominent contributing cause. On the other hand, measles, scarlet fever, erysipelas, poliomyelitis, chorea and several other conditions were less common among the Negroes. Dublin feels that the high death rate among Negroes is due more than anything else to ignorance, poverty, and lack of proper medical care.

Little has been written regarding the disease peculiarities of the Negro infant. Mitchell (60) in 1891 addressed the Woman's Alumnae Society, Woman's Medical College, Philadelphia, on diseases among colored children. Knox and Powers (48) found that a marked reduction in the mortality of infants may be brought about by infant welfare work. The reduction in mortality was more striking for Negro than for white infants and affected particularly the death rate from diarrhea.

It appears that endocrine disorders are less common among Negroes than among whites. Diabetes and exophthalmic goiter (64) have been reported to be less common in Negroes than in whites although the diabetes death rate is rising rapidly in urbanized Negroes (18). In the survey made by Hoffman (39) the admission rate for diseases of the ductless glands and spleen at Johns Hopkins Hospital, 1892-1911, was 0.7 and 2.4 respectively for white males and white females, and 0.1 and 0.3 for colored males and colored females. The lower incidence of endocrine disturbances in Negroes may be a racial characteristic or it may be related to the mode of life, the proportion of intellectual workers being much lower among the Negro than the white population.

The American Negro is to a considerable extent the product of a racial mixture. In this report the term Negro is used to include Mulat-

toes as well as full blooded Negroes. The term "colored" is used in the same sense as in the U. S. Birth and Mortality Statistics to include Negroes, Indians, Chinese, Japanese, and "other colored."

GEOGRAPHICAL DISTRIBUTION

In 1920 Negroes constituted about 10 per cent of the population of the United States (30). Eighty-five per cent of the Negro population live in the Southern States. Since 1790 there has been a slight but steady decrease in the percentage of the Negro population living in the South. Since 1919 this decrease has been more rapid.

Of the Negro population 34 per cent are living in urban communities in comparison with 53 per cent for whites. In the South the Negro lives largely in rural communities, only 25 per cent living in cities of 10,000 inhabitants or more. In the North and West, however, from 75 to 85 per cent reside in urban communities.

ECONOMIC STATUS

The economic status of the Negro population in the United States is, on the whole, much lower than that of the white. From an intensive study of births and deaths in 8 American cities Woodbury (88) concludes that the high infant mortality among the colored population is due largely to a comparatively low economic status. Of the colored births 81.9 per cent occurred in families with an annual income of less than $650 in comparison with 39.6 per cent of the white births.

The proportion of colored mothers employed away from home during and after pregnancy is high. This is an important factor in infant mortality even after allowance has been made for other economic factors (88).

TYPE OF FEEDING

Woodbury (88) studied the type of infant feeding according to race or nationality in 8 Northern cities (U. S. A.). Some of the results of this study are summarized in Table 1. The proportion of infants completely breast fed is slightly higher for whites than for Negroes; the proportion partially breast fed is considerably higher for Negro infants. At the end of the sixth month of life 71 per cent of white infants are completely or partially breast fed in comparison with 77 per cent for Negro infants.

TABLE 1

The Type of Feeding in White and Colored Infants (data from Woodbury, General Table 72, p. 216)

COLOR OR NATIONALITY OF MOTHER	PERCENTAGE OF INFANTS AT THE END OF THE SIXTH MONTH			ARTIFICIALLY FED
	Breast Fed			
	Total	Completely	Partially	
White	70.6	51.9	18.7	29.4
Native	67.1	50.7	16.4	32.9
Foreign Born	75.2	53.5	21.7	24.7
Italian	84.6	64.7	19.9	15.4
Jewish	86.8	56.7	30.1	12.9
French-Canadian	48.7	32.1	16.6	51.3
German	74.8	50.3	24.5	25.2
Polish	86.7	62.2	24.5	13.1
Portuguese	61.8	39.3	22.5	38.3
Colored	77.1	47.8	29.3	22.9

ILLEGITIMACY

Illegitimacy is from 8 to 9 times as common among Negroes as among whites.

BIRTH RATE

The birth rate for Negroes is considerably higher than for whites (Negro, 27.4, white 22.2, U. S. Birth Registration Area, 1924). The proportion of mothers under 20 years of age is high, as is the proportion of mothers having single children.

BODY BUILD IN THE NEGRO

Hrdlička (44) found that Negro children show more uniformity, white children more diversity in their normal physical characters. This becomes more marked as age advances. The head shape of the Negro is less variable than that of the white and is usually long and narrow. The forehead is narrow, prognathism is more common, the malar brows are more prominent and the nose low and broad. The palate of the Negro child is more regular, and the uvula shorter. The teeth are stronger and healthier and irregularities are uncommon. Cutting of the various teeth is more regular. The ears of the Negro are smaller than

those of the white child. There is less adipose tissue and muscular development is better. The weight is smaller but the strength is greater. The chest is deeper, and the pelvis is tilted forward making the buttocks appear prominent. The penis is longer and the Negro female before puberty is more boyish in body build than the white girl.

Schultz (76) found that the Negro fetus can be distinguished from the white as soon as human form is discernible, and that the criteria for differentiation are practically identical with those in later life. According to Schultz the hip breadth in the Negro fetus is narrower in relation to the shoulder breadth than in the white. The forearm in relation to the upper arm is longer and the upper extremity is longer in relation to the trunk and the lower extremity. The hand is narrower and the thumb broader. The lower extremity is longer in relation to the trunk and the leg is longer in relation to the thigh. The foot breadth of the Negro fetus is narrower in relation to the foot length than in the white. The heel is frequently prominent and the great toe is rarely longer than the second toe, a phenomenon not infrequent in whites. The face height in relation to the head height is greater in Negro than in white fetuses. The nose is very broad and the lips thick. The upper lip is higher in relation to the upper facial height than among whites.

PHYSIOLOGICAL DIFFERENCES

According to Riesenfeld and his co-workers (65) there is no difference in the inorganic phosphorus content of placental serum in white and Negro new-borns. The concentration of calcium in the serum of colored new-borns (7a) is slightly but significantly lower than in whites (colored $10.82 \pm .062$, white $11.09 \pm .043$ mg. per cent) (Table 2). This is not dependent on the lower birth weight of Negro infants since no relationship was found between birth weight and calcium concentration of placental serum. There is no difference in the blood pressure of white and Negro new-borns (71). Smillie and Augustine (79) found that the vital capacity of Negro children was lower than that of whites. The difference was less marked when the vital capacity was calculated from stem length than from total height. Roberts and Crabtree (69) found no significant difference in the vital capacity of Negro and white children when calculated from stem length. Vital capacity calculated from total height was lower for Negroes.

According to Woodruff (89) Negroes react to external temperature differently from whites. Black bodies absorb and radiate heat more

TABLE 2

The Influence of Race on the Concentration of Calcium in the Serum of New-Borns

	NUMBER OF CASES	AVERAGE AND PROBABLE ERROR *mg. per cent*	STANDARD DEVIATION AND PROBABLE ERROR *mg. per cent*	COEFFICIENT OF VARIATION *per cent*
All Infants	300	11.00 ± .036	0.92 ± .025	8.4
White Infants				
Total	204	11.09 ± .043	0.91 ± .030	8.2
Male	96	11.09 ± .063	0.92 ± .045	8.3
Female	108	11.08 ± .059	0.91 ± .042	8.2
Colored Infants *				
Total	96	10.82 ± .062	0.90 ± .044	8.3
Male	41	10.94 ± .086	0.81 ± .061	7.4
Female	55	10.73 ± .086	0.96 ± .062	8.9

* Negro and Porto Ricans

Difference in calcium concentration (white minus colored) = 0.27 mg. per cent.
Probable error of difference = 0.075 mg. per cent.
Difference divided by probable error of difference = 3.6.

rapidly than white bodies. Whether or not a body absorbs or radiates heat depends on the difference between the body temperature and the external temperature, heat going from the hotter to the cooler body. It is natural, therefore, Woodruff states, that black individuals will fare better than whites if the external temperature is below body temperature since they will radiate more heat than whites; on the other hand, they are worse off when the external temperature is above body temperature since they absorb more heat than whites.

Schmidt (75), using a mirror and a thermocouple, determined the amount of radiant energy reflected by skins of different colors. He found that a black individual reflected less radiant energy (infra-red through blue) than did white individuals.

Eijkman (19) covered the bulbs of two thermometers with double layers of brown and white skin and exposed them to the sun. The temperature recorded by the thermometer with brown skin on the outside was higher than when the white skin was on the outside.

Martin (58) investigated the amount of heat absorbed by skins of various colors. White skins absorbed from 55 to 65 per cent of the sun's energy, depending on the amount of pigmentation, while Negro skins absorbed 84 per cent.

Schilling (74) studied the influence of skin color and heat absorption in rabbits. A thermometer was inserted under a shaven area of the skin, the animal was exposed to sunlight and the temperature recorded. When the skin was covered with black cotton or was blackened with burnt cork the subcutaneous temperature rose considerably.

Glogner (27) (28) using various types of arm calorimeters, found that the European loses heat less readily than does the Malay.

It appears that Negroes tolerate high external temperatures better than do whites, but it is not altogether clear whether this is a racial characteristic or whether it depends on environmental factors such as acclimatization or training. Negroes tolerate stoke-room temperatures better than whites. Thus the body temperature of a group of whites working in a stoke-room on the Red Sea rose to 39° C, while the temperature of the Negroes varied only a few tenths of a degree from normal (49). Dark skinned individuals are also less susceptible to sun and to heat stroke (49) (22) than are whites.

According to Stigler (81) the rise in body temperature during work at high external temperature is less marked for the Negro than for the white and cooling is more rapid. He does not believe that the better mechanism for regulating body temperature on the part of the Negro is due to a reduction in metabolic rate since the rise in body temperature is the same for the races when heat elimination is interfered with, e. g. in a hot water or steam bath. Nor is it dependent on a larger amount of perspiration since perspiration is excessive in both races. He believes that the better heat regulation is probably due to greater heat loss from radiation and conduction and attributes it to the greater number and larger size of the vessels of the Negro skin (11). He does not believe the influence of skin pigment sufficiently large to play any part in the differences in heat regulation.

Dodd and Wilkinson (15) described a group of infants in whom fever was associated with high summer temperatures. Of seven infants in a ward, five who were white had fever during a hot summer spell, while two Negroes remained fever-free. When the white infants were removed to a cool room their temperature fell to normal and remained there. These cases are of interest but the number is not sufficiently large to be of significance until further extended.

The skin of the Negro is less permeable to ultra-violet radiant energy than white skin (56). Hess (35) found that, using the same dose of ultra-violet radiant energy, rickets could be prevented in white rats but

not in black. Bakwin and Bakwin (7) showed that, when large enough doses of ultra-violet were used, the rate of rise of the serum calcium was the same for white and for Negro infants with tetany. When the dosage was decreased, however, a level was reached at which the serum calcium rose much more rapidly in the white than in the Negro infants, indicating that the minimal amount of ultra-violet necessary for maintaining the serum calcium of Negro infants is greater than for white infants.

DEVELOPMENT DIFFERENCES

Hess and Weinstock (36) observed that, quite apart from the factors of sex or of weight, new-born Negro infants show calcification of the radial epiphyseal centers much more frequently than white infants. They believe that this represents a racial difference. Stettner (80), however, found that environmental factors, such as residence in urban or rural communities, may influence the development of epiphyseal centers in later infancy and it is not inconceivable that prenatal environmental factors may exert an influence on the rate of ossification of the centers.

According to Baldwin (8) and to Dodge (16) the increase in weight of colored infants is slower than that of white infants.

TWIN BIRTHS

The proportion of twin births is considerably higher among colored than among whites (Table 3).

TABLE 3

Living Plural Births (Twins Only) in Per Cent of Total Living Births (U. S. Birth Registration Area)

YEAR	WHITE	COLORED
1918	2.14	2.94
1919	2.26	2.90
1920	2.16	2.66
1921	2.22	2.66
1922	2.18	2.68
1923	2.18	2.71
1924	2.15	2.56
1925	2.10	2.40
1926	2.15	2.50

MOTOR TESTS IN NEGRO INFANTS

Davenport and Steggerda (12) have made Gesell tests on 23 colored infants, 12-13 months of age, in Jamaica where the social environment for the racial groups is more nearly alike than in the United States. According to Gesell, who evaluated these tests, the colored infants were equal to or slightly superior to norms for white children in motor and language development, but somewhat inferior in 6 tests involving adaptations requiring memory and insight.

BLOOD GROUPINGS

Among European and American whites there are from 2.5 to 4.5 times as many individuals in Group II (Group A) as in Group III (Group B) (38). Among African Negroes there are more individuals in Group III than in Group II (38). American Negroes form an intermediate group, the proportion of Group II to Group III individuals being 1.4, a much lower index than for American whites (51).

Landsteiner and Levine (49a) found the "M" substance more frequently in the blood of American Negroes than in that of whites (Negroes, 28.1 per cent; whites, 19.1 per cent). Strong reactions for the substance "P" were about 4 times as frequent in Negro as in white individuals, while negative reactions were rare in the Negro. The "extra agglutinin 1" (49b) was also found more frequently in Negroes than in whites.

PRE-NATAL ENVIRONMENT

Employment away from home during pregnancy is more common among Negro than among white mothers. In the study made by Woodbury (88) the proportion of premature births was highest among mothers employed away from home. Toxic manifestations such as albuminuria, edema, and hypertension are less common in colored than in white mothers. Ford (24) has found that in mothers in whom toxic symptoms were present the still birth rate was high, while in mothers without such symptoms the rate was low. Colored mothers were an exception to this rule, since, though toxic symptoms among them are comparatively infrequent, the still birth rate is high. She noted a similar relationship regarding premature birth, here again the colored mothers being exceptional with a low incidence of toxic manifestations, but a high percentage of premature births.

The high incidence of syphilis in colored mothers is well known. Syphilis is an important factor in the fetal death rate during the later months of pregnancy.

Abnormal presentation and difficult labor are less frequent among Negro than among white mothers. The pelvis of the Negro mother is narrower and deeper than that of the white. Contracted pelves (67) are about four times as frequent in Negro as in white mothers. Contracted pelves are said to be common among primitive peoples in their native abodes (20). Children born of mothers with contracted pelves are apt to be small. Rachitic pelves are also more common among Negro mothers.

As factors contributing to the higher neonatal mortality in Negro infants, Ford points to the higher incidence of contracted and rachitic pelves, the high incidence of syphilis, the high proportion of births to mothers under 20 years, the high proportion of first born infants and infants weighing less than three thousand grams at birth, and the very high proportion of illegitimate children. As factors tending to lower the Negro neonatal mortality rate are the low incidence of toxic manifestations in the mother, the small number of prolonged labors and abnormal presentations, and the small size of the baby and the softness of the head.

It would seem that, under a proper regime, the Negro during fetal and neonatal life might have fully as good a chance of survival as the white, since the factors leading to a high fetal and neonatal mortality in the Negro infant, such as syphilis, high proportion of young and primiparous mothers, and the high incidence of illegitimacy, are largely preventable, while the low incidence of toxic manifestations in the mothers, the small soft head of the Negro infant and the ease of labor are distinct advantages over the white infant. Another factor in which the Negro infant has an advantage during the neonatal period is the low incidence among them of congenital deformities.

DIFFERENCES AT BIRTH

Proportion of the Sexes

The sex ratio at birth, i. e. the ratio of male to female births, is lower in the United States for Negro than for white infants (Negro, 103.4; white, 105.7, U. S. Birth Registration Area, 1918-1925). It is generally assumed that the low birth sex ratio is due to the high still birth

rate, more males dying before birth than females. The possibility that other factors may influence the birth sex ratio is discussed under "Still Birth Rate."

The percentage of premature births is slightly higher among Negroes than among whites. Woodbury (88) found 50 premature births per thousand live births for whites, and 56 for colored. Prematurity occurred more frequently among infants of native born mothers (61 per thousand births) than among those born to foreign born mothers (36 per thousand births) or to colored mothers. In the group studied by Ford (24) prematurity occurred in 51 per thousand births in white mothers and 61 per thousand births in colored mothers.

According to Riggs (68) the Negro new-born weighs considerably less than the white (Negro new-borns, 3104.8 grams; whites, 3316.9 grams). Birth weights for Negro and white infants born in New York City are compared in Table 4. The differences in weight are striking,

TABLE 4

The Birth Weight of White and Negro Infants (Bellevue Hospital, New York City, 1928-1929)

COLOR	NUMBER OF CASES	AVERAGE *grams*	STANDARD DEVIATION *grams*	COEFFICIENT OF VARIATION *per cent*
White—				
Male	608	3390	502	14.8
Female	609	3251	453	14.0
Negro—				
Male	99	3100	450	14.5
Female	88	2916	475	16.3

the white males weighing, on the average, 280 grams more than Negro males, and the females 335 grams.

Still Birth Rate

The still birth rate for Negroes is much higher than for whites (Negro, 7.7 per hundred living births; white, 3.6, U. S. Birth Registration Area, 1922-1925). This is ordinarily attributed to the higher incidence of syphilis among Negroes. There are, however, other factors which influence the still birth rate, such as season of the year (83), residence in urban or rural communities (84), age of parents (85),

TABLE 5

Trend of Infant Mortality for Various States by Color
(U. S. Birth Registration Area, 1915-1928)

STATE	INFANT MORTALITY RATE													
	1915	1916	1917	1918	1919	1920	1921	1922	1923	1924	1925	1926	1927	1928
U. S. Birth Registration Area	100	101	94	101	87	86	76	76	77	71	72	73	65	69
White	99	99	91	97	83	82	72	73	73	67	68	70	61	64
Colored	181	185	151	161	131	132	108	110	117	113	111	112	100	106
Florida	...	...	...	...	...	...	...	...	...	82	74	75	67	67
White	...	...	...	...	...	...	...	...	...	70	60	62	56	55
Colored	...	...	...	...	...	...	...	...	...	107	105	107	93	95
Illinois	...	...	...	...	...	...	...	76	82	71	73	69	64	64
White	...	...	...	...	...	...	...	75	79	68	70	68	63	62
Colored	...	...	...	...	...	...	...	125	157	141	122	109	105	105
Kentucky	...	...	87	93	82	73	62	69	72	65	71	75	61	70
White	...	...	82	87	77	69	58	64	67	61	67	71	58	66
Colored	...	...	152	191	147	138	110	157	157	119	119	134	109	118
Maryland	...	121	120	140	105	104	94	94	95	86	90	87	82	80
White	...	101	101	124	92	90	81	81	80	76	76	74	68	67
Colored	...	209	201	215	160	164	147	147	155	128	146	137	134	128
Mississippi	...	...	...	...	...	...	68	68	68	71	68	70	67	74
White	...	...	...	...	...	...	53	56	53	55	53	59	55	61
Colored	...	...	...	...	...	...	85	79	82	88	83	81	78	86
New Jersey	...	...	...	...	...	...	74	79	72	70	69	70	61	65
White	...	...	...	...	...	...	71	76	69	67	65	67	58	61
Colored	...	...	...	...	...	...	139	129	124	125	125	122	113	124
New York	99	94	91	97	84	86	75	77	72	69	68	71	59	65
White	98	93	90	95	82	85	74	76	71	68	66	68	57	63
Colored	191	169	176	175	151	159	138	124	121	114	119	132	109	123
North Carolina	...	...	100	102	84	85	75	80	81	82	79	82	79	86
White	...	...	85	85	74	73	66	70	70	70	67	71	66	75
Colored	...	...	133	140	109	113	95	101	106	110	105	107	109	109
Ohio	...	...	92	94	90	83	75	72	75	67	70	76	62	66
White	...	...	91	92	88	81	73	70	72	64	67	73	60	64
Colored	...	...	158	178	157	153	122	111	139	113	127	128	103	113
Pennsylvania	110	114	111	129	100	97	88	88	90	79	82	82	69	72
White	108	113	109	126	98	95	86	86	88	76	80	80	67	70
Colored	184	180	194	226	151	167	134	142	151	138	131	139	112	116
Virginia	...	...	98	103	91	84	79	77	84	78	81	84	76	76
White	...	...	80	86	78	72	68	65	71	66	67	72	62	64
Colored	...	...	137	141	120	110	103	102	115	104	111	111	106	105
Dist. of Columbia	111	106	97	112	85	91	83	85	92	76	87	85	...	...
White	83	83	71	85	67	72	68	64	71	62	67	67	...	...
Colored	173	158	160	188	132	139	122	134	143	108	132	123	...	...

order of birth (86), racial stock of parents (62) (53) and employment of mother during pregnancy. In animals diet is an important factor in the still birth rate (31) (32) (33) (21) (57).

The sex ratio for still births is the same for Negroes as for whites, (136 males to 100 females, U. S. Birth Registration Area, 1922-1925). In the study made by Schultz (77) sex ratios for the two racial groups were compared according to the fetal age. In this series the variations for the sex ratio of Negro and white fetuses at various age periods are practically the same.

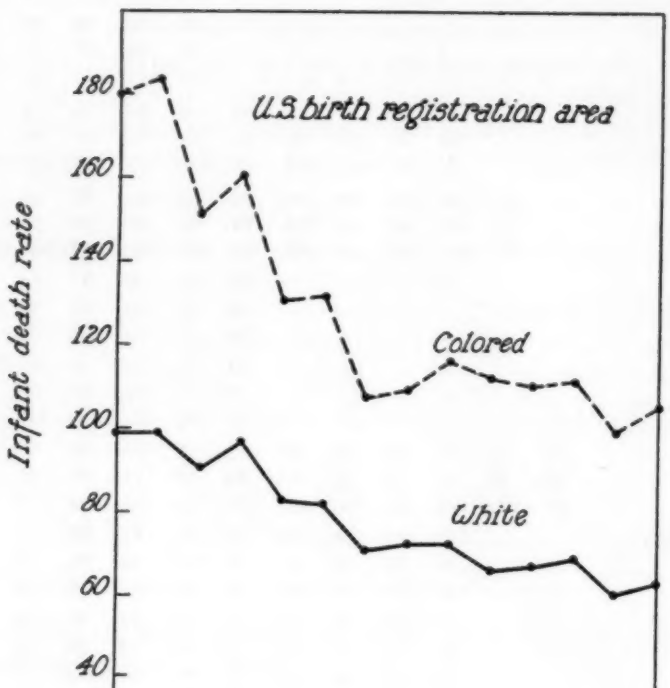

CHART I. THE INFANT MORTALITY RATES FOR COLORED AND WHITE INFANTS

Little (53) has pointed out that, whereas in white matings the sex ratio, i. e. the proportion of males to females, for the first births, including still births, is higher than that for subsequent births, in Negroes the reverse is true, the sex ratio for subsequent births being higher than that for first births.

TREND OF INFANT MORTALITY

In Table 5 and Chart 1 the trends of infant mortality for colored and white infants are compared for the years 1915-1928 (U. S. Birth Registration Area). In evaluating figures for the infant death rate it is important to keep in mind that birth registration in Negro infants is much less satisfactory than in whites and death rates correspondingly less accurate.

In Chart 2 the death rates for colored and white infants are shown in percentage of the average death rates for a 14 year period (1915-1928). The infant mortality for both groups has fallen. The fall has been as rapid for colored infants as for whites. The mortality is decreasing in the Northern states as well as in the Southern.

Though the death rate for Negro infants has been uniformly higher than for white it compares favorably with the infant death rate of many European countries. Thus the Negro infant death rate for Mississippi in 1926 was 81, which is lower than the infant death rate for Belgium (97.4), Denmark (84.4), Hungary (167.5), Finland (85.6), Scotland (83.1), Italy (126.5), and Germany (101.6).

THE MORTALITY RATE BY VARIOUS SUBDIVISIONS OF AGE

In Table 6 and Chart 3 death rates are compared according to various subdivisions of age under one year (U. S. Birth Registration Area, 1918-1925). The Negro infant mortality is higher than the white at all age subdivisions. For infants under one day the difference is not striking; in fact it is not improbable that the slightly higher death rate for Negro infants at this time (7 per cent) is more apparent than real, since birth registration is less nearly complete among Negroes than whites. After the first day the death rate among Negroes is distinctly higher than among whites. The difference is most marked after the first three months of life when the Negro infant mortality rises to about twice that of the white.

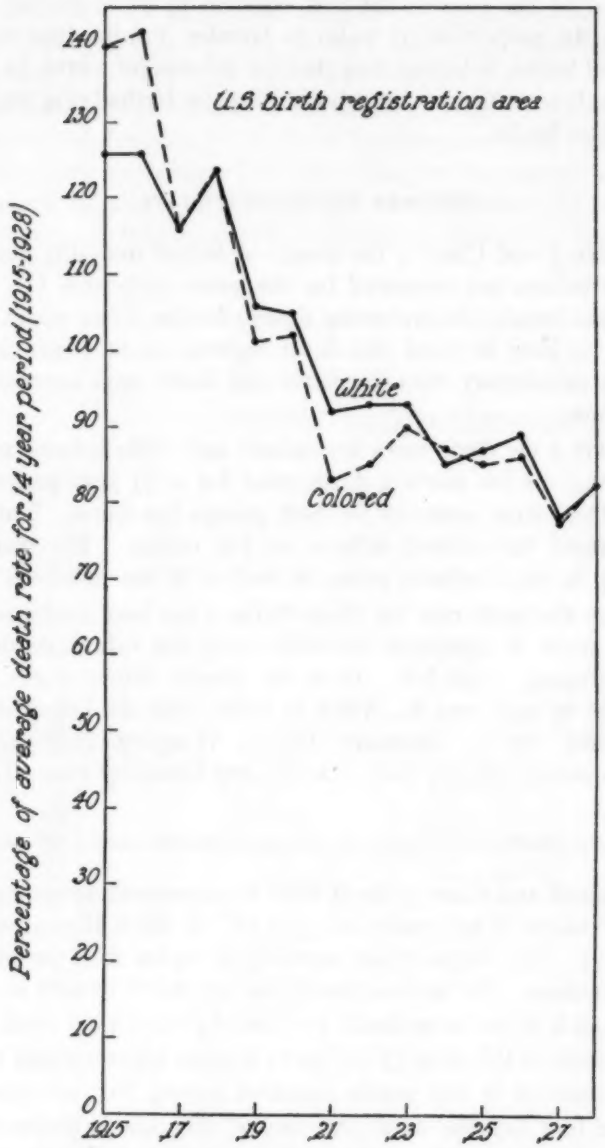

CHART 2. INFANT MORTALITY RATES FOR COLORED AND WHITE INFANTS IN PERCENTAGE OF AVERAGE DEATH RATES FOR A 14 YEAR PERIOD

TABLE 6

Comparison of Infant Mortality in Negro and White Infants by Various Subdivisions of Age (U. S. Birth Registration Area, 1918-1925)

	MORTALITY RATE		NEGRO DEATH RATE IN PER CENT OF WHITE DEATH RATE
	Negro	White	
Under 1 month	53.5	37.3	143
Under 1 day	15.8	14.8	107
1 day	6.0	4.3	140
2 days	4.5	3.3	137
3-6 days	9.5	6.0	159
1 week	8.1	4.8	169
2 weeks	4.6	3.3	140
3 weeks	4.0	2.7	148
1 month	10.2	6.3	164
2 months	8.1	4.9	165
3-5 months	20.4	11.0	186
6-8 months	16.4	8.5	193
9-11 months	12.4	6.7	185

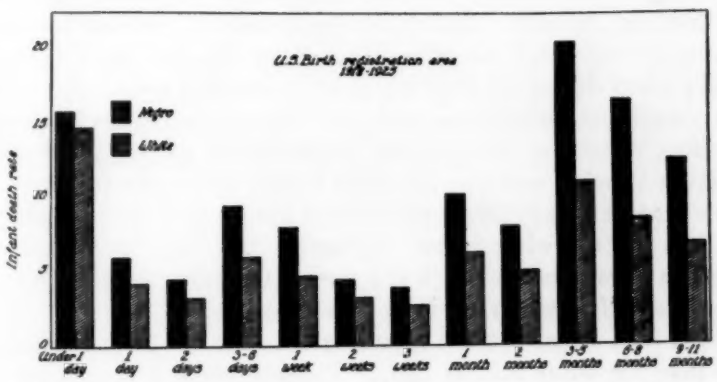

CHART 3. COMPARISON OF NEGRO AND WHITE INFANT DEATH RATES BY VARIOUS SUB-DIVISIONS OF AGE UNDER 1 YEAR

THE MORTALITY SEX RATIO

In Table 7 the mortality sex ratios (i. e. the male death rate in percentage of the female) are compared by various subdivisions of age. At birth the mortality sex ratio for Negro infants is lower than for whites (Negro infants, 125; white infants, 133). This is probably

TABLE 7

Comparison of Mortality Sex Ratio for Negro and White Infants by Various Sub-divisions of Age (U. S. Birth Registration Area, 1918-1925)

AGE	MORTALITY SEX RATIO	
	Negro	White
Under 1 month	128	129
Under 1 day	125	133
1 day	127	132
2 days	139	141
3-6 days	141	128
1 week	117	121
2 weeks	109	121
3 weeks	115	125
1 month	114	132
2 months	119	128
3-5 months	121	124
6-8 months	117	118
9-11 months	111	111

accounted for, in part at least, by the lower incidence of birth trauma among Negro infants, since males are much more frequently injured at birth than are females. During the remainder of the first week the mortality sex ratios for the races do not differ significantly, both showing the marked rise on the third day of life previously noted (3). After the first week the mortality sex ratio for Negro infants falls below that for whites, remaining low until the fourth month of life, after which it is almost identical with that for white infants during the remainder of the first year. It rises in the fourth week of life for the Negro infant just as it does for the white infant. There is, however, no further rise during the second month, but a rise during the third, fourth, fifth and sixth months after which the ratio falls gradually.

It has been stated that the mortality sex ratio for Negro infants is lower than for white infants (40). Though this is true at some age sub-divisions during the first year, it is by no means true for all subdivisions, the mortality sex ratio for Negro infants during the second six months of the first year being practically identical with that for whites, though the death rate is about twice as high during this age period. This is probably accounted for by the very much higher winter death rate from pneumonia among Negroes. It has been shown (3) that there are seasonal variations in the mortality sex ratio, the excess male infant mortality being greater during the winter and spring than during the summer and autumn.

SEASONAL VARIATIONS IN DEATH RATE

In Table 8 and Chart 4 are shown the death rates for white and Negro infants by months of the year. The data are for the Borough of

TABLE 8

The Number of Deaths and the Death Rates of White and Negro Infants From All Causes and From Diarrhea by Months of the Year (Borough of Manhattan, New York City, 1924-1928)

MONTH	NUMBER OF DEATHS				DEATH RATES				NEGRO DEATH RATE IN PERCENTAGE OF WHITE	
	All Causes		Diarrhea		All Causes		Diarrhea		All Causes	Diarrhea
	White	Negro	White	Negro	White	Negro	White	Negro		
ll Months	12,846	2,603	1,638	199	62.3	113.8	8.0	8.7	183	109
nuary	1,172	220	81	12	67.2	113.0	4.7	6.3	168	134
bruary	1,168	263	95	11	72.6	147.2	5.6	5.8	203	104
rch	1,418	297	148	21	81.1	153.5	8.7	11.0	189	127
ril	1,288	291	120	17	75.0	155.2	7.0	8.9	206	127
ay	1,220	235	134	18	70.0	120.6	7.8	9.4	172	121
ne	1,074	207	136	14	63.6	110.5	8.0	7.3	174	92
ly	855	166	116	17	48.8	86.0	6.8	8.9	177	131
gust	954	187	283	29	54.7	95.9	16.6	15.2	176	92
ptember	964	175	221	21	57.1	92.8	12.9	11.0	163	85
tober	929	182	123	13	53.2	94.0	7.2	6.8	177	94
vember	868	184	96	20	50.8	98.5	5.6	10.5	194	188
cember	956	196	85	6	54.6	100.8	5.0	3.1	185	62

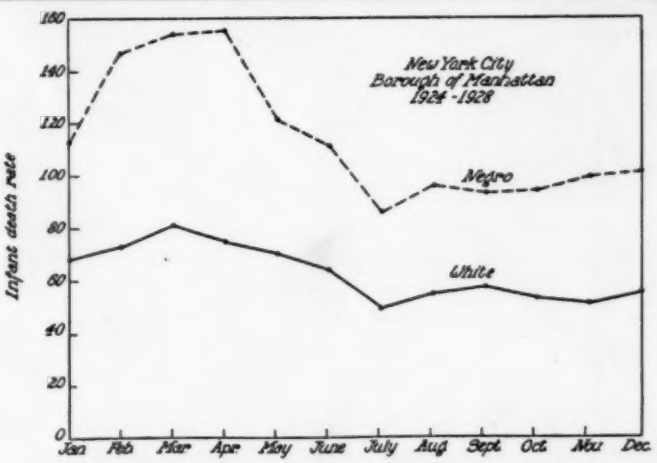

CHART 4. INFANT DEATH RATES OF WHITE AND NEGRO INFANTS BY MONTHS OF THE YEAR

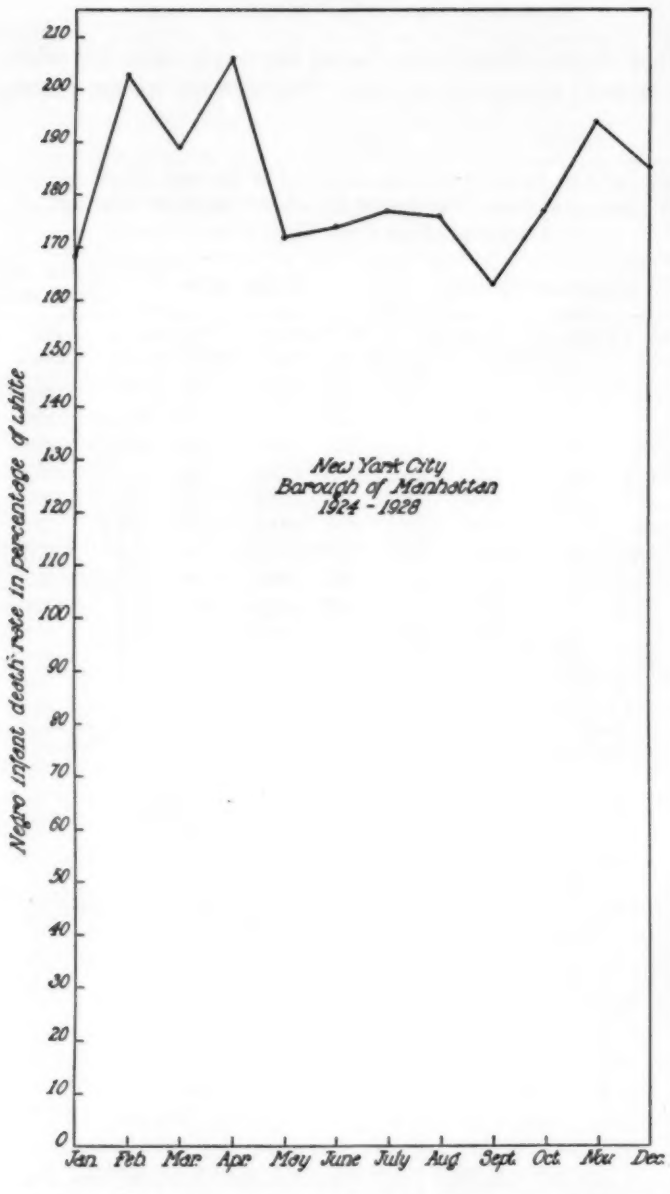

CHART 5. NEGRO INFANT DEATH RATE IN PERCENTAGE OF WHITE

Manhattan, New York City, for the years 1924-1928 and were kindly supplied by Dr. William Guilfoy of the New York City Department of Health. Chart 5 represents the Negro infant death rate by month of the year in percentage of the white. There is a marked seasonal variation in the ratio of Negro to white infant death rates, the excess Negro mortality being considerably greater during the winter than during the summer months.

MORBIDITY AND MORTALITY

Mortality rates for Negro and white infants by various causes are compared in Tables 9 and 10 and Chart 6. It is important to keep in

TABLE 9

Comparison of Death Rates for White and Negro Infants (U. S. Mortality Statistics, 1921-1925)

	CAUSE OF DEATH	WHITE		COLORED	
		Male	Female	Male	Female
	All Causes	89.9	71.8	166.5	140.4
V.	Diseases of the Respiratory System	13.0	10.3	29.0	23.9
VI.	Diseases of the Digestive System	15.8	12.4	26.2	22.3
IX.	Diseases of the Skin and Cellular Tissue	0.4	0.3	0.3	0.2
XV.	Ill Defined Causes	2.3	1.9	22.5	18.8
97 a, b.	Diseases of the Nasal Fossae and Nasal Fossae Annexa	0.1	0.1	1.0	0.8

mind that deaths recorded as due to ill defined causes are ten times as common in Negro as in white infants (U. S. Mortality Statistics, 1921-1925) indicating the less efficient medical care given the Negro child.

According to the U. S. Mortality Statistics, deaths due to diseases of the respiratory system are more than twice as common in Negro as in white infants. Diseases of the digestive system are also more common in Negro infants but not to the same extent as diseases of the respiratory system. Diseases of the skin and cellular tissues are less common in Negro infants.

TABLE 10

Comparisons of Death Rates for White and Negro Infants (U. S. Birth Registration Area, 1918-1925)

DETAILED INT. LIST NUMBER	CAUSE OF DEATH	WHITE			NEGRO			NE DEA RATI PERCE AGE WE
		Total	Males	Females	Total	Males	Females	
	All Causes	76.3	84.4	62.2	121.8	132.4	111.2	180
7	Measles	0.7	0.8	0.7	0.8	0.8	0.7	114
8	Scarlet Fever	0.1	0.1	0.1	0.0	0.0	0.0	...
9	Whooping Cough	2.0	1.9	2.1	4.4	4.3	4.6	220
10	Diphtheria	0.4	0.4	0.3	0.6	0.7	0.6	150
11	Influenza	2.2	2.5	1.9	4.8	5.3	4.3	218
16 113	Dysentery, Diarrhea and Enteritis	13.0	14.3	11.5	18.6	19.7	17.4	143
21	Erysipelas	0.4	0.4	0.4	0.2	0.2	0.1	50
29	Tetanus	0.0	0.0	0.0	0.5	0.6	0.5	...
31 to 37	Tuberculosis, All forms	0.8	0.9	0.8	1.5	1.6	1.4	187
32	Tuberculosis of the Meninges, etc.	0.4	0.5	0.4	0.4	0.5	0.4	100
38	Syphilis	0.6	0.7	0.6	2.9	3.2	2.6	482
80	Convulsions	0.8	0.9	0.6	1.9	2.2	1.6	238
100-101	Pneumonia, all forms	9.9	11.0	8.7	18.4	20.3	16.4	186
159	Congenital Malformations	6.5	7.2	5.8	3.0	3.3	2.6	46
160	Congenital Debility	5.0	5.6	4.3	8.6	9.5	7.7	172
161 a	Premature Birth	18.0	19.8	16.0	23.1	25.0	21.4	128
161 b	Injuries at Birth	4.4	5.3	3.5	2.6	3.2	2.0	59

RICKETS AND TETANY

Severe rickets and tetany are much more prevalent among Negroes than among white infants. The manifestations are similar to those in white children. As stated above the higher incidence among Negroes depends, in part at least, on the lower permeability of the pigmented skin to ultra-violet radiant energy.

It is interesting to note in this connection that Burhans and Smith (9) found that the breast milk of colored mothers contained less calcium

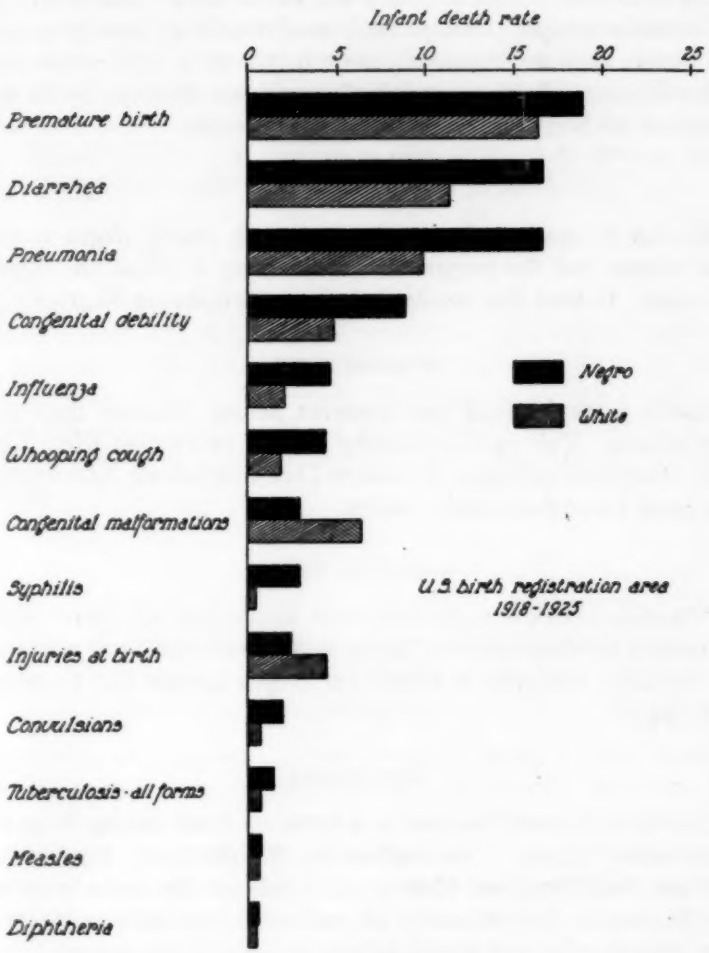

CHART 6. COMPARISON OF DEATH RATES OF WHITE AND NEGRO INFANTS DUE TO VARIOUS CAUSES

and inorganic phosphorus and a higher concentration of chlorides than the milk of white mothers.

Head nodding and nystagmus are more common in Negroes than in whites. Residence in dark houses and streets is an important etiological factor in this condition (63).

Pot belly and umbilical hernia are much more common in Negro than in white infants. Though these conditions may develop in infants with rickets they are frequently seen in young Negro infants before the development of rickets, and furthermore are observed in the native habitats of the Negro where rickets is not prevalent.

MEASLES

Measles is about as often a cause of death among Negro as among white infants and the proportion of the sexes is about the same for both races. In later life measles is less common among Negroes.

SCARLET FEVER

Scarlet fever is much less common among Negroes than among white infants. This racial immunity persists throughout life. Fischer (23a) found the percentage of positive Dick tests among African natives to be much lower than among whites.

WHOOPING COUGH

Whooping cough is more than twice as common as a cause of death in Negro as in white infants (Negro infant death rate, 4.2; white, 2.0). The mortality sex ratio is higher for Negro infants (97.2) than for white (94.3).

DIPHTHERIA

Diphtheria is more common as a cause of death among Negro than among white infants. Observations by Wright (90), Zingher (91), Doull and Bull (17), and Holmes (41) indicate that there is no striking difference in the percentage of positive Schick tests in Negro and white children. In later life diphtheria is less common among Negroes.

SYPHILIS

Syphilis is much more prevalent in Negro than in white infants (Negro infant death rate 2.8; white 0.7). There are no reports indicating racial differences in the disease manifestations such as are seen in the adult.

Jeans and Cooke (45) estimate the incidence of congenital syphilis in St. Louis to be 15 per cent in the Negro race, 1.8 per cent in the

poor of the white race and less than 1 per cent in the well-to-do classes. Since syphilis in the Negro population has been increasing since 1919, it is probable that congenital syphilis is also increasing. Jeans (46) found that 30 per cent of all living births in a syphilitic family die in infancy as compared to a rate of 15 per cent of individuals of the same class without syphilis. He estimates that from 25 to 30 per cent of clinically syphilitic infants die as a result of syphilis.

RHINITIS

Severe non-syphilitic rhinitis occurs more commonly in Negro than in white infants and is perhaps dependent on the nasal configuration. The infant mortality rate from diseases of the nasal fossae and nasal adnexae is 0.1 and 0.1 respectively for white males and females, and 1.0 and 0.8 respectively for Negro males and females. Severe rhinitis in Negro infants is much more frequently observed clinically than in whites.

INFLUENZA

Influenza, like other respiratory infections, is more often fatal in Negro than in white infants (Negro infant death rate, 4.8; white, 2.2).

TUBERCULOSIS

According to Asserson (1) tuberculous infection in New York City is no more common among colored than white infants, the incidence of positive tuberculin reactions among infants admitted to a New York Hospital being 11.3 per cent for whites (3,374 cases), and 11.6 per cent for colored (594 cases). The mortality rate for the Negro infant, however, is much higher than for the white (Negro, 1.6; white, 0.8). Colored children continue to react to the intracutaneous tuberculin test in the same proportion as white through the eighth year, after which the proportion of positive reaction among the colored rises above that of white children (37).

PNEUMONIA

The infant death rate from pneumonia (all forms) is almost twice as high in Negroes as in whites. The greater susceptibility to pneumonia persists throughout life. In spite of the greater frequency of

pneumonia in Negroes, however, there is some reason to believe that the case fatality rate is lower (43) (47) particularly after the fifth decade of life.

TETANUS

The death rate from tetanus is nine times as high among Negro as among white infants.

CONVULSIONS

Convulsions are more than twice as frequent as a cause of death among Negro than among white infants. Convulsions, particularly those severe enough to result in death, are usually due to some grave underlying condition such as organic disease of the brain or meninges, tetany or so-called acute intestinal intoxication. The greater recorded frequency of convulsions as a cause of death in Negro infants may be real or may simply represent a higher percentage of incomplete diagnoses.

BIRTH INJURY

The death rate for birth injuries is considerably lower in Negro than in white infants, (Negro, 2.9; white, 4.4). This corresponds with the experience of obstetricians, who find difficulty at birth among Negro mothers comparatively uncommon despite the frequency of contracted and rachitic pelves. The low death rate from birth injuries is dependent, in part at least, on the infrequency of abnormal presentations and difficult labors in black mothers and on the small size and softness of the head of the Negro infant.

CONGENITAL MALFORMATIONS

The death rate for congenital malformations in Negro infants is less than one-half that for whites, (Negroes, 3.0; white, 6.5). This difference is probably due, in part, to less careful observation of Negroes. In the study by Hoffman (39), however, at the Johns Hopkins Hospital, a similarly low incidence of abnormalities and congenital malformations was found in Negroes, the admission rate being 0.6 and 0.4 per cent respectively, for white males and white females and 0.3 and 0.2 per cent for Negro males and Negro females. Certain deformities

such as pyloric stenosis, mongolian idiocy, cretinism and achondroplasia are rare in Negroes. On the other hand, sickle cell anemia appears to be limited to the Negro race and acrocephaly and polydactylism are said to be more common in Negroes.

DIARRHEAL DISEASES

The recorded death rate due to diarrhea in Negro infants is about 45 per cent higher than for whites for the U. S. Birth Registration Area. For New York City, Borough of Manhattan, 1924-1928 (Table 8) the death rate from diarrheal diseases for Negro infants was only slightly higher than that for whites, (Negro, 8.7; white, 8.0) although the infant death rate from all causes was considerably higher (Negro 113.8; white, 62.3).*

In the study of causal factors in infant mortality based on investigations in 8 cities reported by Woodbury (88) the death rate from gastric and intestinal diseases was lower for colored than for white infants (colored, 28.1; white, 32.6) although the colored infant mortality from all causes was considerably higher (colored, 154.4; white, 108.3). The death rate from respiratory diseases was more than twice as high among colored infants (colored, 44.6; white, 17.9).

Knox and Powers (48), in a study of the effect of welfare clinics on infant mortality in Baltimore, found that the most striking reduction was brought about in the colored death rate due to diarrheal and nutritional diseases, (Table 11). Whereas the colored rate in the unsupervised group was 23, in the supervised it was only 0.6. The reduction

TABLE 11

The Influence of Infant Welfare Clinics on the Death Rate of White and Negro Infants (Summary of Data of Knox and Powers)

CAUSE OF DEATH	DEATH RATE			
	White		Negro	
	Control	Supervised	Control	Supervised
All Causes	36	18	72	19
Diarrheal and Nutritional Diseases	21	3	23	0.6
Respiratory Diseases	10	8	31	15

* From data kindly supplied by Dr. William Guilfoy, N. Y. City Dept. of Health.

in the white death rate from the same group of diseases was from 21 to 3. It is of interest that the racial difference in diarrheal death rate in the unsupervised group was only slight (white, 21; colored, 23) while the death rate from all causes was twice as high for the colored infants (white, 36; colored, 72). The death rate from respiratory diseases remained high among the supervised colored infants (white, 8; colored, 15).

It appears that, when the climatic factor is kept constant, as indicated by the data for New York City and for the eight American cities reported by Woodbury, there is no significant racial difference in the death rate from diarrheal diseases.

ERYSIPELAS

Erysipelas is less common among Negro infants than among whites. The racial immunity of the Negro persists throughout life.

COMMENT

The only differences in disease incidence which may, at present, be safely attributed to the factor of race (i. e. Negro in comparison with white) are dependent on differences in the skin. The Negro skin is less permeable to ultra-violet radiant energy and this is one factor explaining the greater incidence of rickets and tetany among Negro children in temperate climates. This property of the Negro skin, in so far as it is related to rickets and tetany, may be neutralized by adequate exposure to sunlight (as in sub-tropical or tropical climates), or to ultra-violet radiant energy from artificial sources, or by the administration of vitamin D.

A second difference in the Negro skin is its reaction to external heat. When the external temperature is high, body temperature regulation is better in the Negro than the white individual. This explains, in part at least, their greater ability to withstand high external temperatures, e. g. in stoke rooms, and the lower incidence among them of heat-stroke.

Numerous investigators (72) (87) (2) (23) (26) have shown that a correlation exists between high external temperatures and the incidence of summer diarrhea, the rate rising (or falling) when the external temperature rises above (or falls below) the ordinary summer level, although the factor of external humidity has not been ruled out (26).

It is possible that the more effective mechanism of the Negro for withstanding high external temperatures may account for the fact that, in certain communities (e. g. New York City, 8 Northern cities studied by Woodbury) in spite of a poorer hygienic environment, the Negro infant death rate from diarrheal diseases is no higher than the whites; and for the greater ease with which the diarrhea death rate may be controlled in Negroes (47).†

A third property of the skin, which is probably racial, may account for the comparative immunity of the Negro to various skin infections, such as furunculosis and erysipelas.

BIBLIOGRAPHY

(1) Asserson, M. A. The incidence, significance and course of infection in racial and other groups, and its relation to exposure. *Am. Rev. Tuber.*, 16: 359 (Oct.) 1927.

(2) Baginsky, A. Über den Durchfall und Brechdurchfall der Kinder. *Jahrb. f. Kinderh.*, 8: 310, 1875.

(3) Bakwin, H. The sex factor in infant mortality. Human Biol., 1:90 (Jan.) 1929.

(4) Bakwin, H., and Bakwin, R. M. Body build in infants. I. The technique of measuring the external dimensions of the body in infants. *Jour. Clin. Invest.*, 10: 369 (June) 1931.

(5) ————. II. The proportions of the external dimensions of the healthy infant during the first year of life. *Ibid.*, 10: 377 (June) 1931.

(6) ————. III. Body build in disease. *Ibid.*, 10: 395 (June) 1931.

(7) ————. Dosage of ultraviolet radiation in infants with tetany. *Jour. Am. Med. Assn.*, 95: 396 (Aug. 9) 1930.

(7a) Bakwin, H. and Bakwin, R. M. Factors influencing the calcium concentration in the serum of new-borns. Am. Jour. Hyg. In Press.

(8) Baldwin, B. T. Physical growth of children from birth to maturity. U. of Iowa Studies in Child Welfare, 1:1, 1921.

(9) Burhans, C. W., and Smith, D. N. The inorganic constituents of human milk with particular reference to racial variations. *Am. Jour. Dis. Child.*, 26: 303 (Oct.) 1923.

(10) Cartwright, S. Diseases and physical peculiarities of the Negro race. *N. O. Med. Surg. Jour.*, 7: 691, 1850-1851. *Ibid.*, 8: 187, 1851-1852. *Ibid.*, 8: 361, 1851-1852.

(11) Däubler, K. Grundzüge der Tropenhygiene. Berlin, 1900, O. Enslin.

(12) Davenport, C. B., and Steggerda, M. Race crossing in Jamaica. Carneg. Instit., Wash. Publ. No. 395, 1929.

† There are no recorded studies of the reaction of the Negro to low external temperatures. Such studies would be of interest in relation to the susceptibility of the Negro to pneumonia.

(13) DAVISON, H. M., and THOROUGHMAN, J. C. A study of heart disease in the Negro race. *South Med. Jour.*, 21: 464 (Jun.) 1928.

(14) DAY, G. H. Urological and venereal idiosyncrasies presented by the Negro. *Jour. Urol.*, 5: 19 (Jan.) 1921.

(15) DODD, K., and WILKINSON, S. J. External heat a cause of fever in children. *Jour. Am. Med. Assn.*, 88: 787 (Mar. 12) 1927.

(16) DODGE, C. T. J. Weight of colored infants. *Am. Jour. Phys. Anthropol.*, 10: 336 (Jul.) 1927.

(17) DOULL, J. A., and BULL. Quoted by Holmes (41).

(18) DUBLIN, L. I. The health of the Negro. Ann. Am. Acad. Polit. & Soc. Sci. Phila., Pub. no. 2177, Nov., 1928.

(19) EIJKMAN, C. Vergleichende Untersuchungen über die physikalische Wärmeregulierung bei dem europäischen und dem malaiischen Tropenbewohner. *Virchow Arch.*, 140: 125, 1895.

(20) ENGELMAN, G. J. Labor among Primitive Peoples. St. Louis, 1883, 2nd edition.

(21) EVANS, H. M., and BISHOP, K. S. The production of sterility with nutritional regimes adequate for growth and its cure with other foodstuffs. *Jour. Metab. Research*, 3: 233 (Feb.) 1923.

(22) FIGUERAS, L. Insolacion y acaloramiento. *Siglo Medico*, 68: 286 (Mar. 26) 1921.

(23) FINKELSTEIN, H. Über den Sommergipfel der Saüglingssterblichkeit. *Deutsch. med. Wchschr.*, 35[2]: 1377 (Aug. 12) 1909.

(23a) FISCHER, O. Racial immunity to scarlet fever. Münch. Med. Wchschr. 77: 1749 (Oct. 10) 1930.

(24) FORD, H. W. A statistical study on neonatal mortality with special relation to the factor of mother nativity. *Am. Jour. Hyg.*, 7: 89 (Jan.) 1927.

(25) FOX, H. Observations on skin diseases in the Negro. *Jour. Cutan. Dis.*, 26: 67, 1908. *Ibid.*, 26: 109, 1908.

(26) GAFAFER, W. M. Infant diarrhea and enteritis and climate, London 1876-1927. *Am. Jour. Hyg.*, 11: 535 (May) 1930.

(27) GLOGNER, M. Über einem physiologischen Unterschied der Haut des Europäers und der des Malaien. *Virchow Arch.*, 116: 540, 1889.

(28) ————. Beiträge zu den "Abweichungen vom Physiologischen" bei den in den Tropen lebenden Europäern. *Virchow Arch.*, 119: 254, 1890.

(29) GOLDSTEIN, H. J. Diabetes in the Negro. *Jour. Med. Soc. N. J.*, 19: 157 (June) 1922.

(30) GOVER, M. Mortality among Negroes in the United States. Pub. Health Bull. no. 174, 1928.

(31) HART, E. B., MCCOLLUM, E. V., STEENBOCK, H., and HUMPHREY, G. C. Physiological effect on growth and reproduction of rations balanced from restricted sources. Univ. Wis. Agricult. Exp. Sta. Research Bull. no. 17, June, 1911.

(32) HART, E. B., and STEENBOCK, H. Maintenance and reproduction with grains and grain products as the sole dietary. *Jour. Biol. Chem.*, 39: 209 (Sept.) 1919.

(33) HART, E. B., STEENBOCK, H., and HUMPHREY, G. C. Influence of rations restricted to the oat plant on reproduction in cattle. Univ. Wis. Exp. Sta. Research Bull. no. 49, Nov., 1920.

(34) HAZEN, H. H. Diseases of the Skin. K. Kimpton, London, 1927, 3rd edition, p. 441.

(35) HESS, A. F. Newer aspects of the rickets problem. *Jour. Am. Med. Assn.*, 78: 1177 (Apr. 22) 1922.

(36) HESS, A. F., and WEINSTOCK, M. A comparison of the evolution of carpal centers in white and Negro new-born infants. *Am. Jour. Dis. Child.*, 29: 347 (Mar.) 1925.

(37) HETHERINGTON, H. W., MCPHEDRAN, F. M., LANDIS, H. R. M., and OPIE, E. L. A survey to determine the prevalence of tuberculous infection in school children. *Am. Rev. Tuber.*, 20: 421 (Oct.) 1929.

(38) HIRSCHFELD, L., and HIRSCHFELD, H. Serological differences between the blood of different races. *Lancet*, 2: 675 (Oct. 18) 1919.

(39) HOFFMAN, F. L. The statistical experience data of the Johns Hopkins Hospital, Baltimore, Md. 1892-1911. The Johns Hopkins Hospital Reports Monographs. New Series, no. IV, 1913.

(40) HOLMES, S. J. Sex ratio in infant mortality. Univ. of Calif. Publ. Zoology, 29: 267, 1926.

(41) ————. The resistant ectoderm of the Negro. *Am. Jour. Phys. Anthropol.*, 12: 139. 1928.

(42) ————. Differential mortality in the Negro. HUMAN BIOLOGY, 3: 71 (Feb.) 1931. *Ibid.*, 3: 203 (May) 1931.

(43) HOWARD, D. C., and LOVE, A. G. Influenza—U. S. Army. *The Military Surgeon*, 46: 522, 1920.

(44) HRDLIČKA, A. Physical difference between white and colored children. *Proc. Am. Ass. Adv. Sci.*, 47: 475, 1898.

(45) JEANS, P. C., and COOKE, J. V. A study of the incidence of hereditary syphilis. *Am. Jour. Dis. Child.*, 22: 402 (Oct.) 1921.

(46) JEANS, P. C. Syphilis and its relation to infant mortality. *Am. Jour. Syphilis*, 3: 114 (Jan.) 1919.

(47) KELLY, F. B. Observations on 6500 cases of lobar pneumonia at the Cook County Hospital, Chicago. *Jour. of Infec. Dis.*, 38: 24 (Jan.) 1926.

(48) KNOX, J. H. M., and POWERS, G. F. Effectiveness of infant welfare clinics from a medical point of view. *Jour. Am. Med. Assn.*, 78: 707 (Mar. 11) 1922.

(49) KURRER, A. Über Temperaturerhöhungen bei Heizern. *Deutsche Vierteljahrsschr. f. öffentliche Gesundheitspflege*, 24: 291, 1892.

(49a) LANDSTEINER, K., and LEVINE, P. On the racial distribution of some agglutinable structures of human blood. *Jour. Immunol.*, 16: 123, 1929.

(49b) ————. On the inheritance and racial distribution of agglutinable properties of human blood. *Jour. Immunol.*, 18: 87, 1930.

(50) LEMAN, I. I. Diabetes mellitus in the Negro race. *South. Med. Jour.*, 14: 522 (Jul.) 1921.

(51) LEWIS, J. H., and HENDERSON, D. L. The racial distribution of isohemagglutinin groups. *Jour. Am. Med. Assn.*, 79: 1422 (Oct. 21) 1922.

(52) LIEFMAN, H., and LINDEMAN, A. Der Einfluss der Hitze auf die Sterblichkeit der Säuglinge in Berlin (und anderen Grossstäden). *Deutsche Vierteljahrsschr f. öffentliche Gesundheitspflege*, 43: 333, 1911.

(53) LITTLE, C. C. A note on the human sex ratio. *Proc. Nat. Acad. Sci.*, 6: 250 (May) 1920.

(54) ————. Some factors influencing the human sex ratio. *Proc. Soc. Exp. Biol. & Med.*, 16: 127, 1919.

(55) LOVE, A. G., and DAVENPORT, C. B. A comparison of white and colored troops in respect to incidence of disease. *Proc. Nat. Acad. Sci.*, 5: 58, 1919.

(56) MACHT, D. I., ANDERSON, W. T., and BELL, F. K. The penetration of ultraviolet rays into live animal tissues. *Jour. Am. Med. Assn.*, 90: 161 (Jan. 21) 1928.

(57) MACOMBER, D. Effect of a diet low in calcium on fertility pregnancy and lactation in the rat. *Jour. Am. Med. Assn.*, 88: 6 (Jan. 1), 1927.

(58) MARTIN, C. J. Thermal adjustment of man and animals to external conditions. The Croonian Lectures, *Lancet*, 2: 561 (Sept. 13), 1930. *Ibid:* 617 (Sept. 20), 1930. *Ibid:* 673 (Sept. 27), 1930.

(59) MATAS, R. Surgical peculiarities of the Negro. *Trans. Am. Surg. Ass.*, 14: 483, 1896.

(60) MITCHELL, M. V. Clinical notes from diseases among colored children. *Rep. Proc. Alum. Assn. Woman's Med. College*, 1891.

(61) MORISON, R. B. Personal observations on skin diseases in the Negro. *Med. News*, 53: 439 (Oct. 20) 1888.

(62) PEARL, M., and PEARL, R. On the relation of race crossing to the sex ratio. *Biol. Bull.*, 15: 194, 1908.

(63) RAUDWITZ, R. W. Zur Lehre vom Spasmus Nutans, *Jahrb. f. Kinderh.*, 45: 145, 1897.

(64) REDFERN, T. C. Incidence of exophthalmic goiter in Negroes. (Correspondence) *Jour. Am. Med. Assn.*, 75: 51 (Jul. 3), 1920.

(65) RIESENFELD, A., HANDELMAN, I., and ROSE, A. Inorganic phosphorus in the blood of the new-born. *Am. Jour. Dis. Child.*, 30: 646 (Nov.) 1925.

(66) RIETSCHEL, H. Sommerhitze, Wohnungstemperatur und Säuglingssterblichkeit. *Zeitschr f. Kinderh.*, 1: 546, 1910-11.

(67) RIGGS, T. F. A comparative study of white and Negro pelves, etc. *Johns Hopkins Hospital Rep.*, 12: 421, 1904.

(68) ————. Quoted by J. W. Williams, Obstetrics, 1920, p. 155. Appleton & Co., New York.

(69) ROBERTS, F. L., and CRABTREE, J. A. The vital capacity of the Negro child. *Jour. Am. Med. Assn.*, 88: 1950 (Jun. 18) 1927.

(70) ROSSER, C. Rectal pathology in the Negro. *Jour. Am. Med. Assn.*, 84: 93 (Jan. 10) 1925.

(71) RUCKER, M. P., and CONNELL, J. W. Blood pressure in the new-born. *Am. Jour. Dis. Child.*, 27: 6 (Jan.) 1924.

(72) RUSH, B. Medical Inquiries and Observations. Chap. VI. An inquiry into the causes and cure of the cholera infantum. 1789.

(73) SCHEPPEGRELL, W. The comparative pathology of the Negro in diseases of the nose, throat and ear, from an analysis of 11,855 cases. *Ann. Opthal. Otol.*, 4: 589, 1895.

(74) SCHILLING, C. Tropenhygiene, Leipzig, 1909, p. 175.

(75) SCHMIDT, P. Über die hygienische Bewertung verschiedenfarbiger Kleidung bei intensiver Sonnenstrahlung. *Archiv f. Hygiene*, 69: 1, 1909.

(76) SCHULTZ, A. H. Fetal growth of man and other primates. *Quart. Rev. Biol.*, 1: 465 (Oct.) 1926.

(77) ————. Studies on the sex ratio in man. *Biol. Bull.*, 34: 257, 1918.

(78) SIMMONDS, N. Observations on reproduction and rearing of young by the rat as influenced by diet. *Am. Jour. Hyg.* (Supp.), 4: 1 (Sept.) 1924.

(79) SMILLIE, W. C., and AUGUSTINE, D. L. Vital capacity of Negro race. *Jour. Am. Med. Assn.*, 87: 2055 (Dec. 18) 1926.

(80) STETTNER, E. Über die Beziehungen der Ossifikation des Handskeletts zu Alter u. Längenwachstum bei gesunden u. kranken Kindern von der Geburt bis zur Pubertät. *Archiv f. Kinderh.*, 68: 342, 1920-21.

(81) STIGLER, R. Vergleich zwischen der Wärmeregulierung der Weissen und der Neger bei Arbeit in überhitzten Räumen. *Arch. f. Physiologie*, 160: 445, 1914-15.

(82) STONE, C. T., and VANZANT, F. R. Heart disease as seen in a Southern clinic. *Jour. Am. Med. Assn.*, 89: 1473 (Oct. 29) 1927.

(83) U. S. BIRTH, STILLBIRTH and Infant Mortality Statistics, 1925, Part II, p. 37.

(84) *Ibid.*, 1925, Part II, p. 36.

(85) *Ibid.*, 1925, Part II, p. 40.

(86) *Ibid.*, 1924, p. 28.

(87) VIRCHOW, R. Über die Sterblichkeitsverhältnisse Berlins. *Berl. klin. Wchnschr.*, 9: 597 (Dec. 9) 1872.

(88) WOODBURY, R. M. Causal factors in infant mortality. U. S. Dept. Labor, Child Bureau Publ. no. 142, 1925.

(89) WOODRUFF, C. E. Blonds and brunettes in the tropics. *N. Y. Med. Jour.*, 96: 721 (Oct. 12) 1912. *Ibid.*, 96: 785 (Oct. 19) 1912.

(90) WRIGHT, L. T. The Schick test with especial reference to the Negro. *Jour. Infect. Dis.*, 21: 265, 1917.

(91) ZINGHER, A. Diphtheria prevention work in the public schools of New York City. *Jour. Am. Med. Assn.*, 77: 835 (Sept. 10), 1921.

THE HEREDITARY TENDENCY TO ELIMINATE THE UPPER LATERAL INCISORS

BY ADOLPH H. SCHULTZ

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In the normal permanent dentition of man the medial incisors are larger than the lateral ones in the upper jaw, but smaller than the lateral incisors in the lower jaw. This is clearly indicated by the few figures in the following table which are based upon the data from very extensive series.

The ranges of variations in the breadth and the thickness of the incisor teeth of adult man according to various authors quoted by Martin (1928)

INCISOR	BREADTH (*mm.*)	THICKNESS (*mm.*)
Upper Medial	6.5 — 10.6	6.2 — 8.3
Upper Lateral	5.0 — 8.3	5.0 — 7.8
Lower Medial	3.5 — 6.5	4.9 — 7.7
Lower Lateral	4.2 — 7.2	5.3 — 7.6

As has been shown by Rosenberg (1895), Röse (1906), Bluntschli (1907) and others, there appears to exist an evolutionary trend to reduce the dentition of man in connection with a shortening of the jaws. This process of reduction affects the dental row not indiscriminately at any place, but only at the opposite ends, the last molars and the incisors. It is very significant that this trend toward the elimination of teeth is not evident in all the incisors but that it is restricted to the smaller incisors in each jaw. These, as shown above, are the upper lateral and the lower medial incisors. In these particular teeth one can observe all the different degrees of reduction, leading in extreme cases to mere small pegs or even to the complete lack of one or both incisors. Röse (1906), in very large series totaling almost 15,000 cases, found marked degeneration or complete absence of lateral upper incisors in Scandinavians in 6 per cent, in Central Europeans in 2 to 4 per cent, in Greeks in 1.4 per

cent, but in several colored races in only about 1 per cent of the cases examined. The lack of these incisors occurs somewhat more frequently in women than in men and very much more frequently in the permanent than in the deciduous dentition. Among 1,000 white Americans Hrdlička (1921) noted degenerate upper lateral incisors in 3 per cent of the cases and the congenital absence of one or both lateral incisors in 1.4 per cent of the males and in 3.0 per cent of the females. The same author found clearly degenerate lateral incisors in 2.6 (♂) to 4.4 (♀) per cent of American negroes and in 8.4 (♂) to 4.0 (♀) per cent of Chinese and the complete absence of one or both of these teeth in 1.6 to 1.8 per cent of negroes and in only 0.2 to 0 per cent of Chinese. Neither Röse nor Hrdlička could report a single instance in which the upper medial incisors were noticeably reduced or congenitally absent. Among some primitive human races the lack of these incisors seems to be exceedingly rare if, indeed, it does occur at all. Schwarz (1925), for instance, failed to find a single case among 667 skulls from New Caledonia and the New Hebrides, Campbell (1925) did not see it in large series of Australian aboriginals, and Shaw (1931) looked for it in vain among the Bantu races of South Africa. However, manifestations of a process of reduction in the incisors are by no means restricted to the higher races of man. As shown by Figure 1, they do occur also among other primates in which, as in man, such reductions are seen

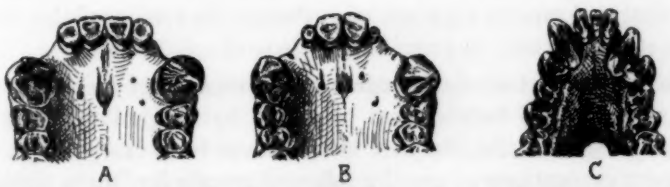

FIG. 1. *A* AND *B* = ANTERIOR PORTION OF UPPER JAW OF ADULT MALE GORILLAS: *A* WITH LATERAL INCISORS OF NORMAL SIZE (P. A. L. 11), *B* WITH VESTIGIAL LATERAL INCISORS (A. S. 760). *C* = ANTERIOR PORTION OF LOWER JAW OF A HOWLER MONKEY (ALOUATTA PALLIATA, A. S. 657) WITH ONE MEDIAL INCISOR CONGENITALLY LACKING

only in the upper lateral and the lower medial incisors. The quite extreme degree of reduction in the lateral incisors of a gorilla, illustrated in Figure 1*B*, is most likely very rare since the author has never seen a similar case among a great many gorilla skulls examined. On the other hand, the complete lack of one medial incisor in the lower jaw

of a howler monkey (Fig. 1*C*) could be found in 5 out of a series of 129 adults of this particular species. Incidentally, none of these same howler monkeys show any sign of reduction in the upper lateral incisors. In contrast to this, in man reductions of the upper incisors are much more frequent than those of the lower incisors.

All these reductions represent really variations in the size of the teeth, ranging from the average proportionate size to the complete disappearance of a tooth. The opposite of the latter extreme variation would, strictly speaking, be represented by an incisor of twice normal relative size. Not even an approach to such a variation has ever been observed. It can be stated, therefore, that one is dealing here with a structure which varies only in one direction, *i. e.*, toward elimination. Such elimination amounts, of course, to a decrease in the number of teeth. The opposite of the latter condition, or an increase in the number of incisors, is known to occur (probably as a reversion to remote ancestral conditions), but three incisors on one side of a jaw are a great deal rarer than only one incisor.

It is a well established fact that many different dental abnormalities and variations are transmitted from one generation to another (see *e. g.* Crosman, 1927). The literature contains a number of more or less complete records showing the hereditary nature of the variations in the size of the upper lateral incisors,[1] including the most extreme variations—the congenital absence of these teeth. It seems desirable to bring these scattered reports together, even though the total available information is still too scanty to permit many general conclusions.

The first report on the hereditary transmission of reduced or eliminated upper lateral incisors was published by McQuillen (1870). As shown by Figure 2, the complete lack of these teeth can occur in several successive generations or may be followed merely by "quite diminutive" or "dwarfed" (McQuillen's terms) teeth in the offspring.

The second report, by Sergi (1914), is of special interest because it demonstrates that in some instances exactly the same degree and form of reduction in the lateral incisors can be inherited through at least three generations. All the members of this family who do show reduced

[1] The writer has never found any published records appertaining to the inheritance of the much rarer, reduced or absent lower medial incisors. He has received, however, incomplete information in regard to a family in which these teeth are congenitally lacking in a number of individuals belonging to at least two different generations.

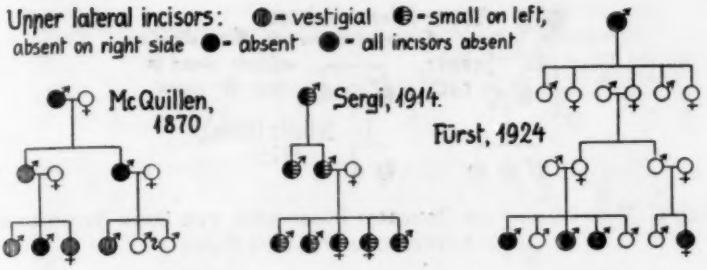
Upper lateral incisors: ◐ = vestigial ◐ = small on left, absent on right side ● = absent ● = all incisors absent
McQuillen, 1870
Sergi, 1914.
Fürst, 1924

FIG. 2. THREE PEDIGREES FROM THE LITERATURE. The two on the left have been constructed from the reports by the respective authors, but are probably incomplete.

teeth have the upper lateral incisor very small on the left and completely lacking on the right side (see Figure 2, middle). Sergi quotes the following additional information:

> Dental agenesia as a phenomenon of heredity has been noted by a number of observers in the past. Hereditary absence of the lateral incisors has been observed in two or three generations and the inheritance may be either direct or alternate. Magitot refers to a woman who showed a lack of the upper, lateral incisors, as did her daughter and nephew; and of a man who lacked the lateral upper right incisor, as did his daughter and his nephew; Leroy d'Etiolle describes the same sort of agenesia in a woman and her three sons. Perrin has cited the case of a family in various members of which the two upper lateral incisors were lacking, and says the defect has existed in the family for many generations.

The next record, by Lenz (1916/18), is unfortunately also incomplete. This author merely states that he knows a family in which the congenital absence of the upper lateral incisors is inherited as "apparently a recessive character."

The pedigree by Fürst (1924) (see Figure 2, right) is most unusual since it contains two cases with the congenital absence of all incisors, an extremely rare condition. This anomaly occurs in the same family in which some members have only the upper lateral incisors missing. Fürst states that the two individuals with absence of "all front teeth" also show an abnormally increased growth of hair. This suggests strongly that in these two cases there existed some general embryonic disturbance affecting both these dermal derivatives—teeth and hair. That tooth defects are often associated with abnormalities of hair has been shown by Parreidt (1886), Reboul (1897) and others.

Figure 3 shows two incomplete pedigrees which demonstrate that the congenital lack of the lateral incisor may be only unilateral in chil-

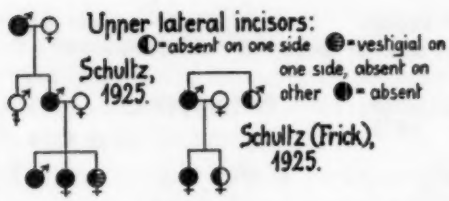

FIG. 3. TWO INCOMPLETE PEDIGREES BASED UPON THE DATA RECORDED BY THE AUTHOR IN A PREVIOUS PAPER

dren of fathers in whom it is bilateral. It can already be concluded that the elimination of the lateral incisors can not only occur in both sexes but can also be transmitted by males as well as by females (see above quotation from Sergi).

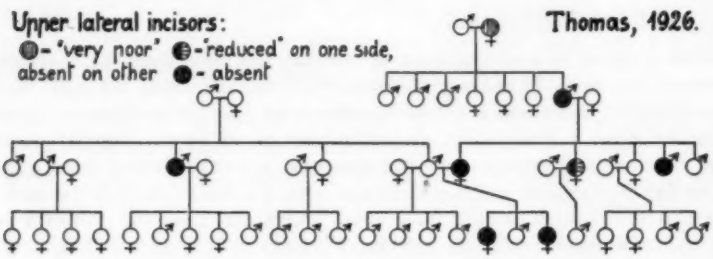

FIG. 4. THE PEDIGREE PUBLISHED BY THOMAS (1926)

Figure 4 gives the extensive and undoubtedly complete pedigree, published by Thomas (1926) based upon records collected by H. R. Hunt. In the large family shown on the left the lateral incisors are normal in all but one individual. Again it is found that a parent with lack of both incisors may have a child in whom only one of these teeth is actually missing, or that a parent with vestigial lateral incisors may have a child in whom both these teeth have entirely disappeared. Thomas concludes that "as far as this case is concerned the results can best be explained by considering the trait as being due to a recessive factor, as the actual results follow the expected results very closely."

The last pedigree, shown in Figure 5, is based upon information which the author was able to collect at the occasion of a large gathering of the particular family (European whites) to be described. The author could himself observe the majority of the individuals. For the missing members of this family he obtained frequently corroborated informa-

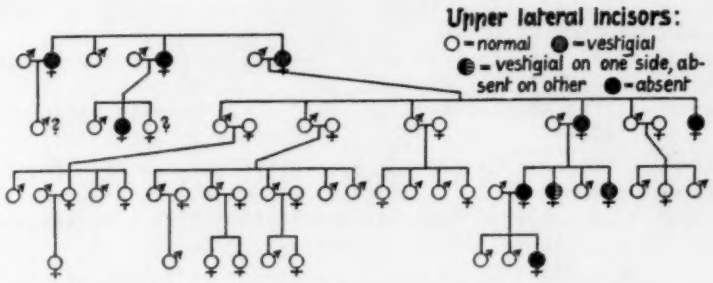

FIG. 5. THE NEW PEDIGREE CONSTRUCTED FROM DATA COLLECTED BY THE AUTHOR IN 1931

tion which appears entirely trustworthy since the informants were fully aware of this "peculiarity in their family." This pedigree is most unusual inasmuch as the character studied is found only in females and is not transmitted by the males. As in some of the previous cases, it is again evident that vestigial lateral incisors in a parent may be followed in the next generation by the complete absence of these teeth, or that the latter condition in a parent may reappear in some children, whereas in others the incisors are merely vestigial or lacking on one side only. One can, therefore, speak only of a hereditary *tendency* toward elimination of the upper lateral incisors, this tendency being manifested in the different cases in varying degrees. In other words, primarily it is not a particular variation, but simply the tendency to vary in the direction of a reduction which is inherited. In this respect these variations in teeth resemble to a certain extent the variations in, *e. g.*, the spinal column which have recently been discussed by Fischer (1931).

As another and final conclusion it can be stated that the tendency to reduce the upper lateral incisors is not genetically alike in all cases, but can be inherited in quite different manners in different families. This is not an exceptional conclusion, but one that has been reached in comprehensive studies of other characteristics as well. Straus (1926), for instance, found very marked diversities in the mode of inheritance of webbed toes in man.

BIBLIOGRAPHY

BLUNTSCHLI, H. 1907. Das Gebiss des Menschen als Zeugniss seiner Vergangenheit. *Wissen und Leben*, I, Zürich.

CAMPBELL, T. D. 1925. Dentition and palate of the Australian aboriginal. *Public., Keith Sheridan Foundation, No. 1, Adelaide.*

CROSMAN, A. M. 1927. Inheritance of abnormalities in teeth. *J. of Heredity*, XVIII, 368-370.

FISCHER, E. 1931. Die Vererbung von Wirbelsäulenvarietäten beim Menschen. *Verhdlg. d. Gesell. f. Phys. Anthropol.*, 5-8.

FÜRST, T. 1924. Der Erbgang der Anodontie. *Arch. f. Rassen- u. Gesellschaftsbiol.*, XVI, 310-311.

HRDLIČKA, A. 1921. Further studies of tooth morphology. *Amer. J. Phys. Anthropol.*, IV, 141-176.

LENZ, F. 1916/18. (Referat über) Kantorowicz, Die Progenie und ihre Vererbung. *Arch. f. Rassen- u. Gesellschaftsbiol.*, XII, 220-221.

MARTIN, R. 1928. Lehrbuch der Anthropologie. *Jena.*

MCQUILLEN, J. H. 1870. Hereditary transmission of dental irregularities. *Dental Cosmos*, XII, 27-29, 73-75, 193-195.

PARREIDT, I. 1886. Ueber die Bezahnung bei Menschen mit abnormer Behaarung. *D. Monatsschr. f. Zahnheilk.*, IV, 41-53.

REBOUL, 1897. Homme velu. *Bull. Soc. Anthropol., Paris.* XXXVII, 444-446.

RÖSE, C. 1906. Ueber die Rückbildung der seitlichen Schneidezähne des Oberkiefers und der Weisheitszähne im menschlichen Gebisse. *D. Monatsschr. f. Zahnheilk.*, XXIV, 225.

ROSENBERG, E. 1895. Ueber Umformungen an den Incisiven der zweiten Zahngeneration des Menschen. *Morphol. Jahrb.*, XXII, 265-339.

SCHULTZ, A. H. 1925. Studies on the evolution of human teeth. *Dental Cosmos*, LXVII, 935-947, 1053-1063.

SCHWARZ, R. 1925. Kiefer und Zähne der Melanesier in morphologischer und morphogenetischer Beziehung. *Schweiz. Monatsschr. f. Zahnheilk.*, XXXV, 43-78.

SERGI, S. 1914. Missing teeth inherited. *J. of Heredity*, V, 559-560.

SHAW, J. C. M. 1931. The Teeth, the Bony Palate and the Mandible in Bantu Races of South Africa. *London.*

STRAUS, W. L., JR. 1926. The nature and inheritance of webbed toes in man. *J. Morphol. & Physiol.*, XLI, 427-439.

THOMAS, L. C. 1926. Five studies in human heredity. *Eugenical News*, XI, 150-153, 155-159.

THE INFLUENCE OF HEREDITY UPON THE DURATION OF LIFE IN MAN BASED ON A CHINESE GENEALOGY FROM 1365 TO 1914*

BY I-CHIN YUAN

The subject of longevity has aroused man's interest since time immemorial and although early records are of doubtful scientific value, our present evidence indicates that man's average duration of life has been increasing within recent times. A large part of this increase has been brought about by the reduction of the death rate in infancy and early childhood. As age advances, the reduction in the death rate becomes less and less marked, and toward the declining years of life, the mortality rate seems in some cases to be actually increasing. This indicates clearly that the study of life duration should be directed toward the determination of the factors which influence man's chance to live out his full life span rather than toward attempts to increase the natural span of life. Such studies will for the most part be statistical in character. Broadly speaking, such statistical inquiries may be classified into two types, one being the demographic analysis of various environmental factors and the other the genealogical investigation of hereditary influence upon the duration of life. The present study is of the second type and in it the writer proposes to examine a Chinese genealogy covering the years from 1365 to 1914 and to see what evidence it contains as to the inheritance of duration of life.

A limited number of studies have been made in which genealogical material has been used in an attempt to evaluate the inheritance of dura-

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* Papers from the Department of Biostatistics, School of Hygiene and Public Health, The Johns Hopkins University, No. 158.

The writer is indebted to Doctor Lowell J. Reed for invaluable help at every stage of this investigation and to Doctor Raymond Pearl for his generous counsel on general biological matters.

tion of life but since these have been reviewed by the writer elsewhere[1] they will not be considered at this time.

In a statistical investigation of this character it is necessary to examine the material critically to determine its limitations and merits for the problem at hand, and the advantages and disadvantages of the genealogy under consideration will therefore be considered in detail.

The source of the present material is a Chinese genealogy obtained through the kindness of Dr. Ting-an Li and from which the writer[2] recently worked out a series of life tables for a period of several hundred years. This record belongs to a southern Chinese family which has lived in the Chung shan district near Canton ever since the first ancestor came there from the northern part of Kwangtung in the latter part of the thirteenth century. The first complete personal account in this genealogy registers 1365 as the year of birth of the ancestor of the fourth generation. From this date, the record gives a continuous family bookkeeping of nineteen more generations up to the year 1914, when the last count of the family was taken.

In this genealogy, the male lineage of the family is traced from the first known ancestor giving a personal sketch of each male, recording the name of the father, the title of social distinction, the dates of birth and death and the place of burial, together with a similar description of his wife or wives including the names of sons and daughters born to each wife. As a rule, those who died unmarried under fifteen years of age were not recorded, probably because they were considered of no importance in the propagation of the family. For this reason, it contains no information of any kind concerning mortality statistics in infancy and childhood nor any complete record of persons who died between fifteen and twenty years of age. This omission and the lack of information of female descendants are the main statistical limitations of the material. On the other hand, experience gained during the past two years in handling this record has convinced the writer of the high degree of accuracy and completeness in the recording of the lineage of male descent and of the dates of birth and death of persons who lived to twenty years of age and over. Practically every male mentioned in this genealogy can be traced as to his ancestral lineage for every generation,

[1] Yuan, I.-C. A critique of certain earlier work on the inheritance of duration of life in man. *Quart. Rev. Biol.*, (In press).

[2] Life tables for a southern Chinese family from 1365 to 1849. HUMAN BIOLOGY, Vol. 3, No. 2, May, 1931, pp. 157-179.

both in the individual records and in the general summary of pedigrees for the entire family. The recording of the dates of birth and death gives the exact hour, day, month and year of occurrence. This is not surprising to anyone who is familiar with the Chinese tradition of placing great emphasis upon family lineage and the common superstition that one's future is predestined largely by one's hour, day, month and year of birth. It seems, therefore, that this type of genealogy furnishes unusually suitable material for the study of the influence of heredity upon the duration of life.

To facilitate the compilation of data, kinship cards were punched from records of individuals of known relationship. On the left-hand side was recorded the history of an individual and on the right, that of some related individual, the columns between these two records being used to designate their kinship. Desired information was then obtained by sorting and tabulating these kinship cards.

In this paper, the scope of study is limited to one of parental influence upon the duration of life. The first criterion on the basis of which this particular group of data was assembled was that only persons born prior to 1820 were entered as suitable material, since in any consideration of duration of life it is essential that every individual have the opportunity of completing his life span. This limitation allows even the group born in the year 1819 a chance to live to ninety-five years of age, since the last family count was taken in 1914. Very few would have remained alive subsequent to that year, considering the fact that the extreme span of life is about one hundred years. In other words, it eliminates any artificial discrimination as to the chance to live to full length of life.

The second criterion was to exclude anyone who died under twenty years of age, partly because the genealogical record contains no information for those dying under fifteen and partly because the recording of persons dying between fifteen and twenty is probably not as complete as for those of twenty and over, since every Chinese in the old days would have been married by twenty years of age and in a position to propagate the family. It follows therefore that in all comparisons between parent and offspring the individuals concerned must have attained twenty years of age in order to be included in the study.

Table 1 shows the total number of cases available for study for each of the possible combinations of parent and offspring. The frequency distributions of fathers, mothers and sons by year of birth and age at death are given in Tables 2 and 3.

TABLE 1

Total Numbers of Fathers, Mothers and Sons, and of Sons whose Fathers', Mothers' or Fathers' and Mothers' Ages at Death were Known

FATHERS, MOTHERS AND SONS WHO LIVED TO AGE 20 AND OVER	NUMBER
Fathers whose sons' ages at death were known	1,007
Mothers whose sons' ages at death were known	1,160
Sons whose fathers' or mothers' ages at death were known	2,311
(a) whose fathers' ages at death were known	2,144
(b) whose mothers' ages at death were known	2,112
(c) whose fathers' and mothers' ages at death were known	1,945

TABLE 2

Distribution of Individuals by Time of Birth

FREQUENCY DISTRIBUTIONS OF FATHERS, MOTHERS AND SONS BY YEAR OF BIRTH				NUMBER AND PERCENTAGE DISTRIBUTION OF SONS, AND OF MALES OF WHOM THE SONS WERE A SAMPLE				
Year of Birth	Fathers	Mothers	Sons	Year of Birth	Sons Who Lived to Age 20 and Over		Males Who Lived to Age 20 and Over	
					Number	Per cent	Number	Per cent
Before 1400	4	3	2	1365-1599	231	10.00	303	11.09
1400-1449	14	12	14	1600-1699	368	15.92	437	16.00
1450-1499	37	37	45	1700-1749	402	17.39	492	18.01
1500-1549	47	53	82	1750-1799	919	39.77	1026	37.55
1550-1599	66	65	88	1800-1819	391	16.92	474	17.35
1600-1649	95	94	154					
1650-1699	136	156	214					
1700-1749	292	323	402					
1750-1799	316	416	919					
1800-1819	0	1	391					
Totals	1007	1160	2311		2311	100.00	2732	100.00

The imposing of these conditions necessarily reduces the available data to a considerable extent, especially when the family population increases rapidly in time. But the advantage of having homogeneous material avoids undesirable complications in the analysis and interpretation of results.

The next point in the discussion of the material is to inquire as to whether it constitutes a representative sample of the original universe from which it was drawn and also as to how its statistical characteristics compare with those of data usually encountered in the field of vital statistics.

TABLE 3

Distribution of Individuals by Age at Death

AGE IN YEARS	FREQUENCY DISTRIBUTIONS OF FATHERS, MOTHERS AND SONS BY AGE AT DEATH			NUMBER AND PERCENTAGE DISTRIBUTIONS, BY AGE AT DEATH, OF SONS, AND OF MALES, OF WHOM THE SONS WERE A SAMPLE			
				Sons Born During 1365-1819		Males Born During 1365-1819	
	Fathers	Mothers	Sons	Number	Per cent	Number	Per cent
20-24	12	26	98	98	4.24	121	4.43
25-29	33	28	142	142	6.14	166	6.08
30-34	44	44	134	134	5.80	161	5.89
35-39	38	76	143	143	6.19	159	5.82
40-44	64	75	185	185	8.01	219	8.02
45-49	86	68	212	212	9.17	234	8.57
50-54	109	86	212	212	9.17	244	8.93
55-59	109	98	215	215	9.30	268	9.81
60-64	110	112	220	220	9.52	272	9.96
65-69	133	107	242	242	10.47	296	10.83
70-74	90	156	199	199	8.61	214	7.83
75-79	86	129	148	148	6.40	180	6.59
80-84	62	85	106	106	4.59	135	4.94
85-89	21	52	40	40	1.73	43	1.57
90-94	9	11	13	13	.56	17	.62
95-99	1	6	1	1	.04	2	.07
100 & over	0	1	1	1	.04	1	.04
Totals	1007	1160	2311	2311	99.98	2732	100.00

In pairing father and son and mother and son, the necessary condition as already stated was that their age at death not only was known, but that it was at least twenty years. If failure to record age at death is not associated with any particular group of persons, as is probably the case with this material, there should be no differentiation of sons from the general male population or of parents from the general parent group. The requirement of living at least to age twenty probably has a selective effect on the parents because it excludes a particular group of parents whose sons never lived to age twenty. But it should not make any appreciable difference to the sons to exclude parents dying under age twenty since only a small proportion of persons would have become parents under twenty years of age and a still smaller proportion who were parents before age twenty would have died before that age. Since all males are sons and the condition of pairing parent and son should not

for any important reason differentiate sons from the male population, one should expect the distributions by year of birth and age at death of the sons closely to parallel those of the total male group. This is exactly what one actually finds if one examines Tables 2 and 3. The percentage distributions of the sons and of the male population both by year of birth and age at death can hardly be considered significantly different from each other. The year of birth is regrouped partly to obtain larger numbers and partly to compare with the groups of males for whom the writer previously constructed a series of life tables showing the time trend of mortality.

One will also notice from these distributions that the total male group numbers 2,732 and the sons 2,311, constituting about 85 per cent of the total. In other words, only 15 per cent of the males of known age at death are left out of the original material because their parent's age at death was unknown. Moreover, from Table 1 one can show of 2,311 sons, 92.8 per cent (2,144 cases) whose fathers' ages at death were known, 91.4 per cent (2,112 cases) whose mothers' ages at death were known and 84.2 per cent (1,945 cases) whose fathers' and mothers' ages both were known. It may be pointed out in passing that the similar values of the first two percentages (92.8 and 91.4) indicate almost no differentiation in the recording of age at death of the fathers and of the mothers in respect of the sons.

Nothing can be gained by comparing the parents with the general population, because parents are a selected class that one expects to differ from the general population. The comparison of these parents with the general parent group will not be helpful for they were a particular group of parents whose sons lived at least to age twenty. However, in view of the fact that the sons represent a large majority of the male population and have been shown to be undifferentiated from the general male group as to the recording of their ages at death, it is hardly conceivable that their parents should be particularly affected by the same procedure.

The material is sampled from a continuously growing population for the period of 1365-1819 and it will be futile to try to compare it with other data for the same period of time because neither genealogical records nor official vital statistics for such a long interval have been as yet placed on record. One will have to be content with a discussion of the general statistical characteristics of the material as compared with those usually encountered in the field of vital statistics. Considering the

age distribution of deaths of the sons, we see that the curve is one which rises slowly in the adult age period, reaching its modal point just before age seventy, and then decreases rather rapidly to zero at about one hundred years of age. The general sweep of the distribution is one familiar to vital statisticians. Its difference in small details from the death curves for the occidental peoples in recent years is not surprising in view of the racial diversity and the marked difference in time.

The same thing may be said of the age distribution of deaths of fathers and of mothers. Both curves resemble that of the sons in general shape, except that in the case of fathers and mothers the proportion of deaths is lower in earlier ages and higher in later years of life. Although very little is known about the mortality behavior of parents as a class, from general experience we would expect that they would be more selected people since they have been able to rear their children while their fellows have failed and, naturally, more in the parent group will survive to older ages than in the general population. The comparison of the mortality of fathers and mothers shows that fewer mothers than fathers died in middle life, which results in a difference in the modal point of deaths, the mode for mothers being at age seventy to seventy-four and for fathers at age sixty-five to sixty-nine. The latter is again in agreement with the usual finding of a greater number of females than of males surviving to old age. Thus the material exhibits the general statistical characteristics to be found in mortality records of population.

The life table is the most satisfactory device for the analysis of age distributions of mortality. It enables one to present the essential facts of mortality in terms of three important functions, the life table death rate, survivorship, and expectation of life. It is therefore natural that this method should be chosen in an attempt to demonstrate quantitatively the influence of heredity upon the duration of life. The method used in this paper is one developed for constructing an abridged life table and is the same as that employed by the writer in a previous paper.[3] (Details of the method will therefore be omitted from this paper.)

The constants of life tables will differ in their significance according to the type of raw data on which they are based. The population and mortality figures from which the life tables in this study are determined are obtained by actually following a group of people from age twenty

[3] Life tables for a southern Chinese Family from 1365 to 1849. Reference cited.

to their death. This material differs in type from the census counts on which the government life tables are based, and from the exposure group from which insurance life tables are built up.

The life tables obtained from census counts and mortality registrations for any year give an accurate determination of the risk of dying at any age for that particular year. The expectation of life computed from such tables is, however, a purely hypothetical figure, stating the average age of death which would obtain if a population were subjected to these particular age specific death rates throughout their life span. This figure will not agree with the mean after-lifetime of the actual population since the mortality rates for any year do not persist unchanged. For such life tables, therefore, we may consider that the mortality rates represent actual conditions for the census year, but the expectation of life is a theoretical figure which will not agree with observed conditions.

For the life tables of this study the situation is just reversed. Here the mortality rates are averages over a period of more than five centuries and consequently do not describe mortality conditions at any specific time. The average expectation of life, on the other hand, is the average number of years actually lived by these people, and consequently is the most meaningful of the life table constants obtained.

The basic material used by an insurance company results in life tables which occupy an intermediate position between the two just considered. It is composed of a group of people exposed to risk of dying over a period of years, thus differing from the instantaneous group obtained by the census method, but the individuals in the group are continually changing and are not followed continuously until death, so that we do not have a complete record of one group as in the genealogical study. The death rates obtained from these data are averages over a long time period and the expectation of life is a weighted average of the duration of life of the dead and the expectation of life of the living.

The agreement between the expectation of life computed from the life tables and the mean after-lifetime obtained directly from the age distribution of deaths for the population group of this study is shown in Table 4. These figures agree very closely, no difference being greater than .5 year. The slight differences are due to the fact that the mean after-lifetime figures were not smoothed; otherwise the two constants would be identical. The large numbers on which these figures were

TABLE 4

Expectation of Life, $\mathring{e}_x$, Obtained from Life Tables Compared with Mean After-lifetime Calculated Directly from Age Distributions of Deaths

AGE IN YEARS	FATHERS		MOTHERS		SONS	
	Expectation of Life ($\mathring{e}_x$)	Mean after-life-time	Expectation of Life ($\mathring{e}_x$)	Mean after-life-time	Expectation of Life ($\mathring{e}_x$)	Mean after-life-time
20	39.6	39.3	41.3	41.2	34.9	34.9
30	31.0	30.9	33.3	33.0	28.2	28.3
40	23.4	23.3	26.0	26.3	22.0	21.9
50	17.1	17.0	19.4	19.9	16.5	16.7
60	12.2	12.1	13.8	14.0	11.7	11.8
70	8.6	8.3	9.1	8.6	7.7	7.8

based give them greater stability than will be found in later cases where less extensive material was available.

The life table describes and summarizes the mortality experience in a population group so that the age mortality characteristics of different groups can easily be compared with each other. This clearly answers precisely the purpose of the present study in ascertaining the relationship of heredity to the duration of life, since it enables us to compare the age of dying of groups differing as to the length of life of parents.

In studying the influence of heredity on the duration of life by the method described above, the following relationships were examined: (1) the extent to which the mortality characteristics of sons at any particular age differed from each other when sons were classified according to the duration of life of their parents; (2) the amount of difference between the mortality characteristics of the parents at a specific age when parents were classified according to the duration of life of their sons; (3) the quantitative relationship between the duration of life of sons and of their parents. These relationships were studied by means of life tables computed by the method referred to above, for groups of sons whose parents had lives of short, intermediate, and long duration, and for groups of parents whose sons were short, intermediate and long lived. The three divisions of duration of life are defined as age periods at death of 20-49, 50-69, and 70 and over. Arbitrary as they may be, they are not in disagreement with common concepts of length of life. This classification also serves the purpose of dividing the available data

TABLE 5

Life Tables for Fathers, Mothers and Sons

AGE IN YEARS	FATHERS WHOSE SONS LIVED TO AGE 20 AND OVER			MOTHERS WHOSE SONS LIVED TO AGE 20 AND OVER			SONS WHOSE FATHERS OR MOTHERS LIVED TO AGE 20 AND OVER		
	Number alive at beginning of age x	Probability of dying in five-year interval for each one alive at beginning of age x	Expectation of life for each one alive at beginning of age x	Number alive at beginning of age x	Probability of dying in five-year interval for each one alive at beginning of age x	Expectation of life for each one alive at beginning of age x	Number alive at beginning of age x	Probability of dying in five-year interval for each one alive at beginning of age x	Expectation of life for each one alive at beginning of age x
x	l_x	$_5q_x$	$\mathring{e}_x$	l_x	$_5q_x$	$\mathring{e}_x$	l_x	$_5q_x$	$\mathring{e}_x$
20	1000	.016	39.6	1000	.023	41.3	1000	.046	34.9
25	984	.024	35.2	977	.031	37.2	954	.055	31.5
30	960	.036	31.0	947	.039	33.3	902	.067	28.2
35	926	.053	27.1	910	.050	29.6	842	.081	25.0
40	877	.075	23.4	864	.064	26.0	774	.100	22.0
45	811	.104	20.1	809	.083	22.6	697	.123	19.1
50	727	.140	17.1	742	.108	19.4	611	.153	16.5
55	625	.185	14.5	662	.141	16.5	518	.191	14.0
60	510	.239	12.2	568	.183	13.8	419	.240	11.7
65	388	.300	10.2	464	.239	11.3	318	.302	9.6
70	271	.369	8.6	353	.313	9.1	222	.384	7.7
75	171	.442	7.2	242	.410	7.0	137	.490	6.0

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into groups having large enough numbers to give meaningful comparisons of their life table functions.

For the purpose of later comparisons it is desirable to show first life tables for fathers, mothers, and sons independently of each other. These life tables are constructed from mortality distributions as presented in Table 3, giving values of l_x, ${}_5q_x$, and e_x° at each quinquennial age from 20 to 75. The results are shown in Table 5 and Figures 1, 2

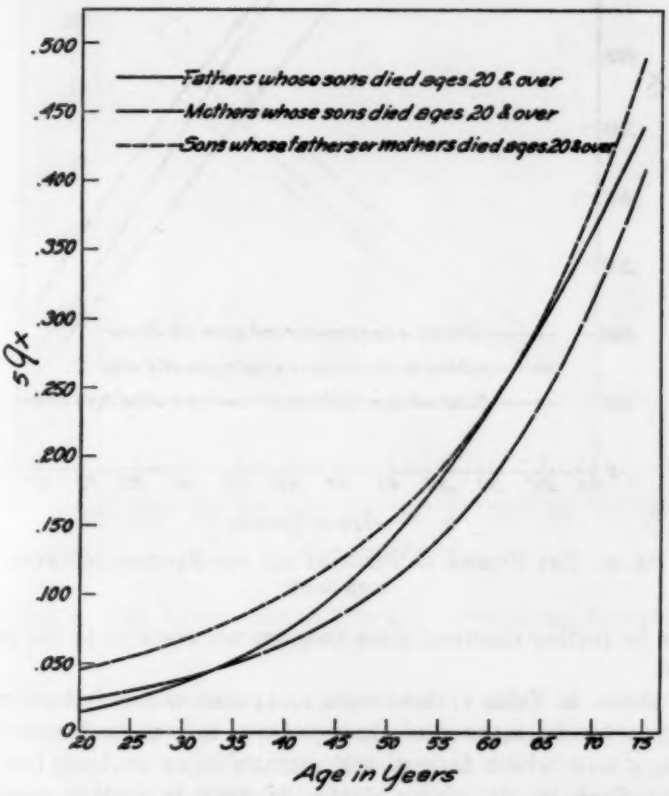


FIG. 1. THE PROBABILITY OF DYING IN FIVE-YEAR INTERVALS (${}_5q_x$) FOR FATHERS, MOTHERS AND SONS

and 3. The life tables clearly exhibit more favorable mortality conditions for each of the parents than for the sons, and the difference is greater between mothers and sons than between fathers and sons. The reasons for these differences have been briefly pointed out before and

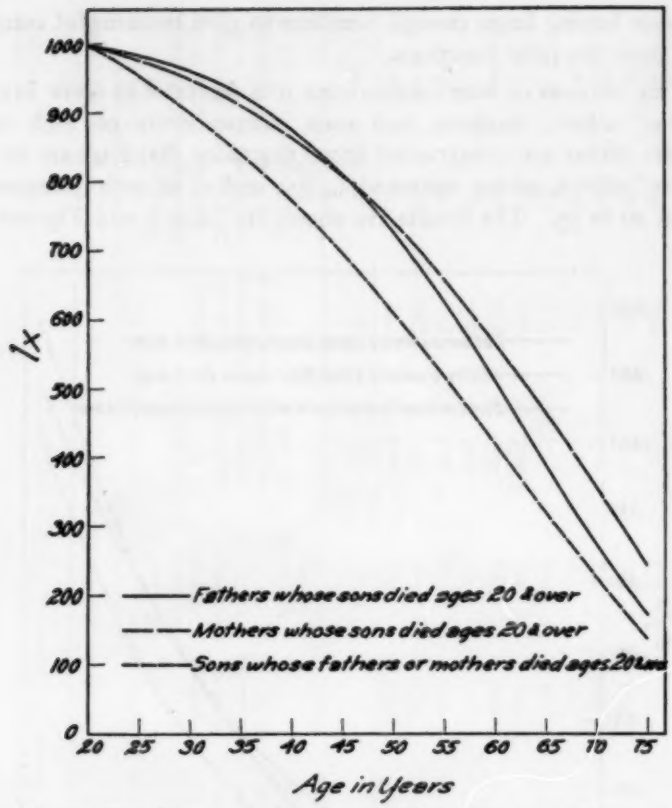


FIG. 2. THE NUMBER OF SURVIVORS (l_x) FOR FATHERS, MOTHERS, AND SONS

will not be further discussed since they are not essential to the problem at hand.

As shown in Table 1, there were 2,144 sons whose fathers' ages at death were known, 2,112 sons whose mothers' ages at death were known and 1,945 sons whose fathers' and mothers' ages at death both were known. Each of the above classes of sons is further subdivided according to their parents' duration of life. The life tables for these different groups of sons will be discussed in the following paragraphs.

On subdividing the sons according to age of death of father, we find that the numbers of sons whose fathers died in the age periods 20-49, 50-69, and 70 and over were 490, 993 and 661 respectively. Their life tables are shown in Table 6 and Figure 4.

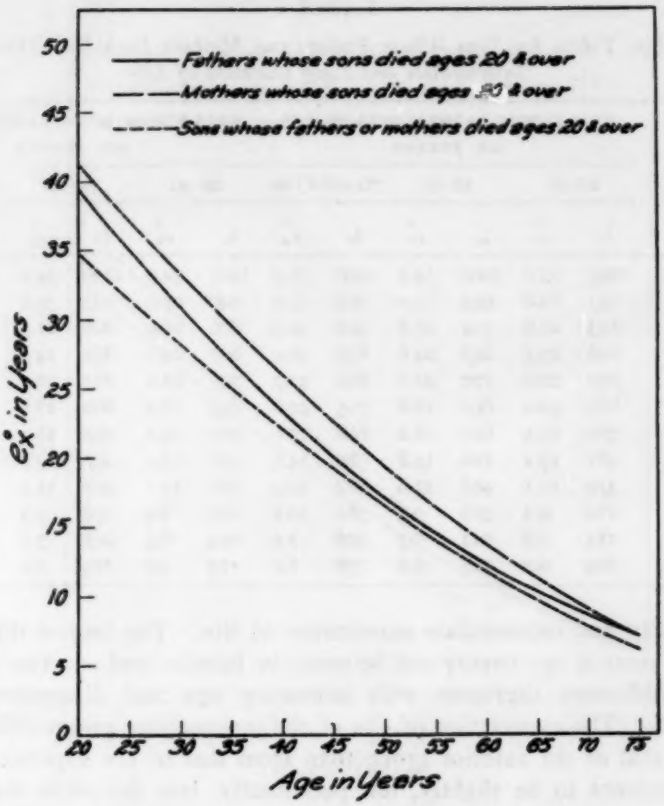


FIG. 3. THE EXPECTATION OF LIFE (e_x°) IN YEARS FOR FATHERS, MOTHERS, AND SONS

Let us first consider the function of survivorship, l_x. Out of 1,000 persons starting together at age twenty, the numbers of survivors at age fifty are, 570 for sons of short-lived fathers, 602 for sons of fathers of intermediate life duration and 668 for sons of long-lived fathers. At age seventy-five there are 109, 129 and 176 survivors respectively.

Now, turn to e_x°, the expectation of life shown in Figure 4 which is of special interest in the present study. The finding is entirely in accord with that of the other function. The sons of the short-lived fathers had the poorest expectation of life, the sons of long-lived fathers had the greatest expectation of life and the sons of fathers of intermediate

TABLE 6

Life Tables for Sons Whose Fathers and Mothers Each Had Short, Intermediate and Long Duration of Life

AGE IN YEARS	SONS WHOSE FATHERS DIED IN THE AGE PERIODS						SONS WHOSE MOTHERS DIED IN THE AGE PERIODS					
	20-49		50-69		70 and Over		20-49		50-69		70 and Over	
x	l_x	$\mathring{e}_x$	l_x	$\mathring{e}_x$	l_x	$\mathring{e}_x$	l_x	$\mathring{e}_x$	l_x	$\mathring{e}_x$	l_x	$\mathring{e}_x$
20	1000	33.2	1000	34.5	1000	37.5	1000	32.9	1000	34.1	1000	38.0
25	951	29.8	955	31.0	962	33.9	940	29.9	953	30.7	972	34.0
30	893	26.6	902	27.7	920	30.3	876	26.9	898	27.5	936	30.2
35	826	23.5	841	24.6	870	26.9	807	24.0	835	24.3	891	26.6
40	750	20.6	770	21.6	812	23.7	733	21.1	763	21.4	836	23.2
45	664	18.0	690	18.8	745	20.6	653	18.4	682	18.6	768	20.0
50	570	15.5	602	16.2	668	17.6	567	15.8	593	16.1	687	17.1
55	471	13.2	506	13.8	580	14.9	476	13.4	497	13.7	594	14.4
60	370	11.1	406	11.6	484	12.4	381	11.1	397	11.5	489	11.9
65	272	9.3	306	9.5	381	10.1	285	8.9	298	9.5	377	9.7
70	183	7.6	211	7.7	276	7.9	194	6.9	205	7.7	266	7.7
75	109	6.0	129	6.1	176	6.0	115	4.9	125	6.0	165	6.0

longevity had intermediate expectation of life. The largest difference is 4.3 years at age twenty and between the inferior and superior groups. The difference decreases with advancing age and disappears after seventy. The expectation of life of the intermediate group differs less from that of the inferior group than from that of the superior group, and appears to be slightly, but persistently, less favorable than that of the sons as a whole, which has been shown in Table 5.

There were 500, 751 and 861 sons whose respective mothers had short, intermediate and long duration of life. Life tables for the three groups of sons are shown in Table 6 and Figure 5. What has been said in discussing the life tables for different groups of sons in respect of their fathers' life duration is exactly applicable to the comparison of the life functions of sons in respect of mothers' life duration. The rate of mortality is highest in sons of short-lived mothers, lowest in sons of long-lived mothers and intermediate in sons of mothers of intermediate longevity. Out of the original groups of 1,000 persons at age twenty, there are surviving 567, 593 and 687 at age fifty, and 115, 125 and 165 at age seventy-five respectively in the three different groups of sons, indicating definitely more favorable survivorship in sons of longer-lived mothers. A comparison of the expectation of life leads

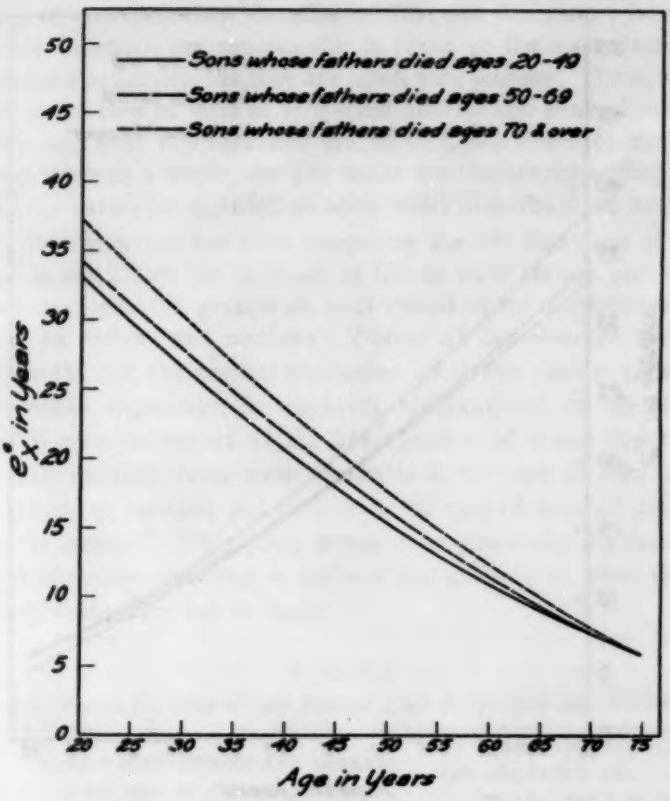


FIG. 4. THE EXPECTATION OF LIFE ($\mathring{e}_x$) IN YEARS FOR SONS WHOSE FATHERS HAD SHORT, INTERMEDIATE AND LONG DURATION OF LIFE

to the conclusion, entirely harmonious with the other facts just brought out, that where mothers were short-lived, sons had the shortest expectation of life, where mothers were long-lived sons had the longest expectation of life and where mothers died in the intermediate period of life sons had expectation of life intermediate to the other two. In general, the differences of these life table functions are smaller between sons of short-lived mothers and sons of mothers of intermediate duration of life than between sons of long-lived mothers and sons of mothers of intermediate duration of life.

The next logical step is to consider whether or not there is any real difference between the life tables for sons classified by age at death of

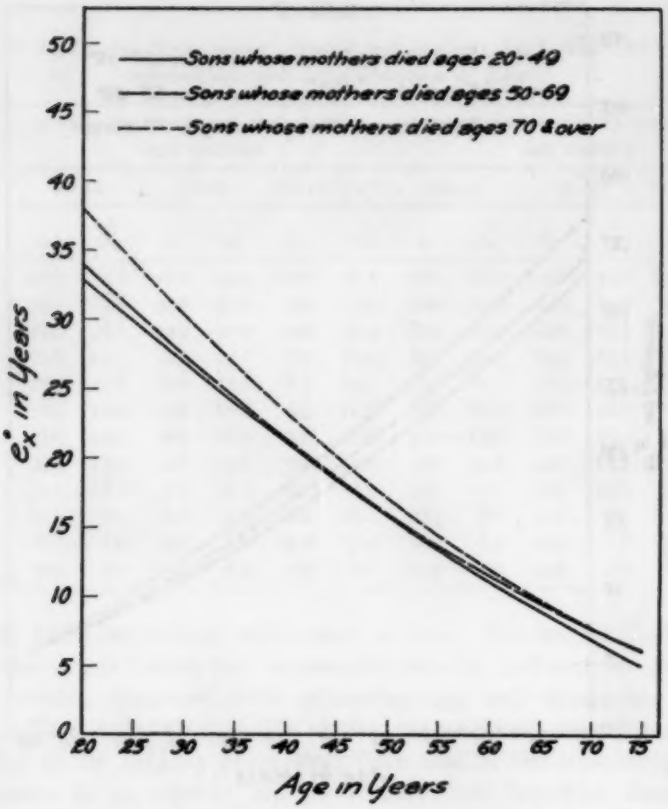


FIG. 5. THE EXPECTATION OF LIFE ($\mathring{e}_x$) IN YEARS FOR SONS WHOSE MOTHERS HAD SHORT, INTERMEDIATE AND LONG DURATION OF LIFE

fathers and for sons classified by corresponding age at death of mothers. First, compare the life functions for sons of short-lived fathers with those for sons of short-lived mothers. The death rate of sons of short-lived mothers is higher in earlier ages, lower in middle life and tends to become less favorable again in later life than the corresponding death rates for sons of short-lived fathers. Consequently, their survivorship is relatively poorer in earlier life and better in the middle years and their expectation of life is relatively better in middle life than either earlier or later years. However, all these differences are very small. In the case of sons of fathers of intermediate longevity and of sons of

mothers of corresponding duration of life, the differences in each of these life functions are consistently in favor of the sons classified by their fathers' longevity, but they are again very minute. The same thing is seen in the case of sons of long-lived fathers and sons of long-lived mothers, and their life functions are, in fact, still closer to each other. Viewing them as a whole, the life tables for the corresponding groups of sons can hardly be regarded as being really different from each other.

So far, the writer has been comparing the life functions of groups of sons in respect of the duration of life of their fathers and of their mothers and also the groups of sons classified by corresponding life duration of fathers and mothers. Taking all the evidence which has been brought out, the general conclusion up to this time is plainly that the mortality experience is markedly differentiated in the different groups of sons in respect of the life duration of either their fathers or of their mothers, being most favorable in the case of sons of long-lived fathers or mothers and poorest in the case of sons of short-lived fathers or mothers. This effect seems to be practically the same when sons are classified according to fathers' age at death as when classified according to mothers' age at death.

TABLE 7

Life Tables for Sons Whose Parents Died at Specified Age Periods

AGE IN YEARS	SONS WHOSE FATHERS AND MOTHERS BOTH DIED IN THE AGE PERIODS			SONS WHOSE PARENTS DIED ONE IN ONE AGE PERIOD AND THE OTHER IN ANOTHER AGE PERIOD		
	20-49	50-69	70 and Over	20-49 & 50-69	20-49 & 70 and Over	50-69 & 70 and Over
x	$\mathring{e}_x$	$\mathring{e}_x$	$\mathring{e}_x$	$\mathring{e}_x$	$\mathring{e}_x$	$\mathring{e}_x$
20	31.5	33.6	39.8	32.7	35.4	37.3
25	28.3	30.0	35.7	29.5	31.8	33.5
30	25.4	26.7	31.8	26.5	28.3	29.9
35	22.6	23.5	28.0	23.5	25.0	26.5
40	20.0	20.6	24.4	20.7	21.9	23.3
45	17.5	17.8	21.0	18.0	19.0	20.3
50	15.2	15.3	17.8	15.4	16.4	17.5
55	13.1	13.0	14.8	13.0	14.0	15.0
60	11.2	10.9	12.1	10.7	11.8	12.7
65	9.4	9.0	9.7	8.6	9.8	10.6
70	7.8	7.4	7.6	6.8	8.0	8.8
75	6.4	5.8	5.7	5.0	6.5	7.1

Having seen definite evidence of what influence one parent, without regard to the other parent, can have upon the mortality behavior of the sons, we shall now examine the combined influence of both parents. Accordingly, sons whose fathers' and mothers' ages at death both were known are grouped in respect of the life duration of both parents. Out of 1,945 sons of the above category, there were 131, 327 and 324 cases whose fathers and mothers both died in age periods of 20-49, 50-69, and 70 and over respectively, and 376, 252 and 535 cases one of whose parents died in one age period while the other died in another age period in the combinations of 20-49 and 50-69, 20-49 and 70 and over, and

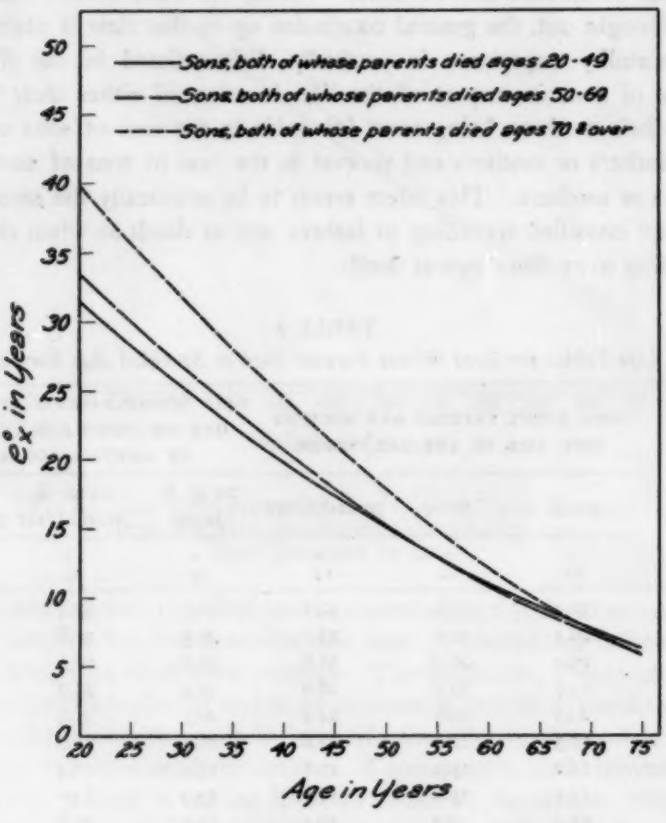


FIG. 6. THE EXPECTATION OF LIFE (e°_{x}) IN YEARS FOR SONS WHOSE FATHERS AND MOTHERS BOTH HAD SHORT, INTERMEDIATE AND LONG DURATION OF LIFE

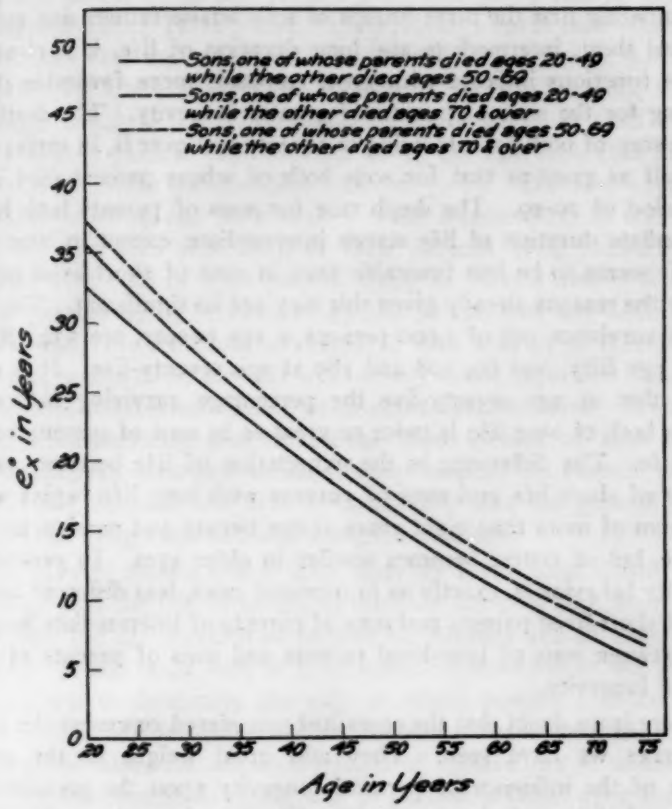

Sons, one of whose parents died ages 20-49 while the other died ages 50-69
Sons, one of whose parents died ages 20-49 while the other died ages 70 & over
Sons, one of whose parents died ages 50-69 while the other died ages 70 & over
e°_{x} in Years
50
45
40
35
30
25
20
15
10
5
0
20 25 30 35 40 45 50 55 60 65 70 75
Age in Years

FIG. 7. THE EXPECTATION OF LIFE (e°_{x}) IN YEARS FOR SONS ONE OF WHOSE PARENTS DIED IN ONE PERIOD OF LIFE DURATION WHILE THE OTHER DIED IN ANOTHER PERIOD OF LIFE DURATION

50-69 and 70 and over. The life functions for each of these six groups of sons are shown in Table 7 and in Figures 6 and 7.

It may help in the discussion below to point out now the fact that the foregoing grouping necessarily divides the material into smaller units upon which the computations are based. They are, however, of comparable size and, except for one group in which only 131 cases are available, will serve with sufficient margin of safety the present purpose of an abridged partial life table. The only caution necessary is to keep in mind that the life functions become less reliable in older ages as they are necessarily more affected by small numbers.

Comparing first the three groups of sons whose fathers and mothers both had short, intermediate and long duration of life, it is clear that the life functions indicate with great emphasis more favorable chance of living for the sons of parents of greater longevity. The death rate for the sons of both parents living to seventy and over is, in earlier ages, only half as great as that for sons both of whose parents died in the age period of 20-49. The death rate for sons of parents both having intermediate duration of life stands intermediate, except in later years when it seems to be less favorable than in sons of short-lived parents, but for the reasons already given this may not be significant. The numbers of survivors, out of 1,000 persons at age twenty, are 534, 583 and 731 at age fifty, and 99, 108 and 189 at age seventy-five. It is worth noting that at age seventy-five the percentage surviving in sons of parents both of long life is twice as great as in sons of parents both of short life. The difference in the expectation of life between sons of parents of short life and sons of parents with long life begins with a maximum of more than eight years at age twenty and persists throughout life, but of course becomes smaller in older ages. In general, the mortality behavior is, exactly as in previous cases, less different between sons of short-lived parents and sons of parents of intermediate longevity than between sons of long-lived parents and sons of parents of intermediate longevity.

There is no doubt that the cases just considered represent the largest differences we have seen. They add great weight to the general picture of the influence of parental longevity upon the probability of living and dying of the sons. The life functions show the highest mortality of all in the case of sons of parents both dying in the age interval 20-49, and are higher than in the case of sons only one of whose parents was known to have died in that age interval. They are the most favorable of all in sons whose parents both lived to seventy and over, these, similarly, being more favorable than in sons only one of whose parents was known to have lived to that age. The sons of parents both of intermediate life duration clearly stand in an intermediate position between sons with one of their parents dying between 20-49 and sons with one of their parents dying at 70 and over. This is again in accord with the general run of evidence that sons of parents of intermediate life duration have intermediate mortality risks.

The last groups of sons remaining for comparison are those whose fathers and mothers died in different age periods in combinations of

20-49 and 50-69, 20-49 and 70 and over, and 50-69 and 70 and over. An examination of Table 7 and Figure 7 brings forth once more clear-cut evidence that the matter of living and dying is highly selective in favor of sons whose parents lived longer. The life functions for sons of parents living to 20-49 and 50-69 are more favorable than for sons both of whose parents died between 20-49 but less favorable than for sons both of whose parents died between 50-69. The sons of parents who lived to 50-69 and 70 and over stand intermediate in the risk of dying between sons with both of their parents dying between 50-69 and sons with both of their parents dying at 70 and over. While the sons of parents dying between 20-49 and 70 and over are distinctly inferior in their life functions as compared with sons of parents living to 50-69 and 70 and over, they are without any doubt superior as compared with sons of short-lived fathers or of short-lived mothers. From these results, it is perfectly clear that these three groups of sons are not only themselves arranged in an orderly manner in respect of their parents' life duration but also fit beautifully into the general picture as if each of the groups had a preassigned position.

The general results which have been set forth point clearly to the conclusion that the life functions in the sons are to a high degree associated with the life duration of fathers, of mothers and of both parents together. It will be of interest to examine the converse of the previous question and to determine the way in which parents classified as to sons' duration of life, differ in their mortality characteristics. In constructing life tables for different groups of fathers and of mothers, the frequency distribution in each group of parents is based upon the actual number of fathers and of mothers whose sons died in the age period 20-49, 50-69 or 70 and over. Each parent is entered only once in any particular group even if he had two or more sons dying in that age period. In other words, these life tables are not weighted according to the fertility of parents because fertility and longevity are known to be correlated and the present problem is to determine the difference in longevity of parents in respect of sons' life duration and not of parents' fertility. Table 8 shows the life functions of groups of fathers and of mothers whose sons had respectively short, intermediate and long durations of life.

The life functions for the three groups of fathers in respect of sons' life duration are shown graphically in Figure 8. The death rate is consistently lower for fathers whose sons lived longer. Out of 1,000

TABLE 8

Life Tables for Fathers and Mothers Whose Sons Had Short, Intermediate and Long Duration of Life

AGE IN YEARS	FATHERS WHOSE SONS DIED IN THE AGE PERIODS						MOTHERS WHOSE SONS DIED IN THE AGE PERIODS					
	20-49		50-69		70 and Over		20-49		50-69		70 and Over	
x	l_x	$\mathring{e}_x$	l_x	$\mathring{e}_x$	l_x	$\mathring{e}_x$	l_x	$\mathring{e}_x$	l_x	$\mathring{e}_x$	l_x	$\mathring{e}_x$
20	1000	39.0	1000	40.9	1000	42.7	1000	40.5	1000	43.6	1000	44.6
25	984	34.6	986	36.5	988	38.2	979	36.3	982	39.4	978	40.6
30	959	30.4	965	32.2	970	33.9	951	32.3	960	35.2	953	36.6
35	924	26.5	935	28.2	944	29.7	914	28.5	932	31.2	923	32.7
40	874	22.9	893	24.4	907	25.8	865	25.0	897	27.4	887	28.9
45	807	19.5	835	20.9	856	22.2	803	21.7	852	23.7	844	25.2
50	721	16.6	759	17.7	789	18.9	727	18.7	796	20.2	792	21.7
55	616	14.0	665	14.9	704	15.8	637	16.0	725	16.9	728	18.4
60	498	11.7	553	12.3	601	13.1	535	13.5	637	13.8	651	15.3
65	374	9.7	430	11.1	483	10.7	425	11.4	532	11.1	559	12.4
70	256	8.0	306	8.2	357	8.6	315	9.5	410	8.6	452	9.8
75	156	6.6	193	6.6	235	6.8	214	7.8	278	6.4	332	7.4

alive at age twenty, there are for the respective groups of fathers 721, 759 and 789 survivors at age fifty; and 156, 193 and 235 survivors at age seventy-five. The $\mathring{e}_x$'s in years are 39.0, 40.9, and 42.7 at age twenty; 16.6, 17.7, and 18.9 at age fifty; and 6.6, 6.6, and 6.8 at age seventy-five respectively. In fact, the relative positions of the mortality experience in the different groups of fathers in respect of sons' life duration are quite similar to those of sons in respect of fathers' life duration.

What has just been seen in the comparison of different groups of fathers in respect of their sons' life duration is in general true in the case of different groups of mothers in respect of the life duration of their sons. The mortality changes are such that the expectations of life in years for different groups of mothers are 40.5, 43.6, and 44.6 at age twenty; 18.7, 20.2, and 21.7 at age fifty, and 7.8, 6.4, and 7.4 at age seventy-five. This is not unlike the case of sons classified according to the life duration of their mothers. These life curves are shown in Figure 9.

From the results seen both in the groups of fathers and of mothers, it is not difficult to conclude that the mortality experience is most favorable in parents whose sons lived longest, poorest in parents whose sons

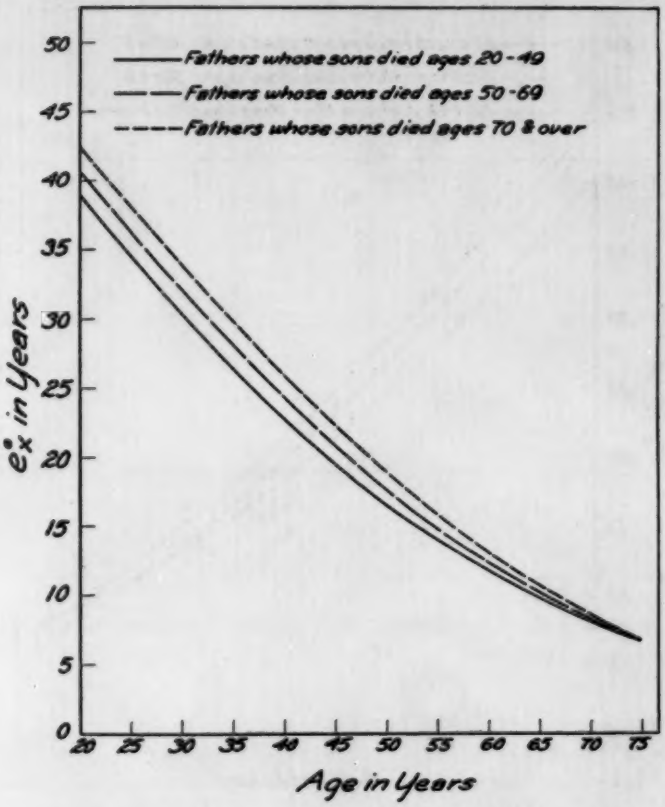


FIG. 8. THE EXPECTATION OF LIFE (e_x°) IN YEARS FOR FATHERS WHOSE SONS HAD SHORT, INTERMEDIATE AND LONG DURATION OF LIFE

lived shortest and intermediate in parents whose sons had an intermediate length of life. This conclusion is exactly the same in character as the one which has been reached from the study of mortality experience in different groups of sons in respect of their parents' life duration. Both of them point to the inference that the chance of living and dying is highly selective according to parental longevity.

The question which naturally arises at this stage of the discussion is as to what quantitative relationship exists between the duration of life of parents and of sons. Owing to the limited material, no divisions of life duration finer than the rather wide intervals of 20-49, 50-69,

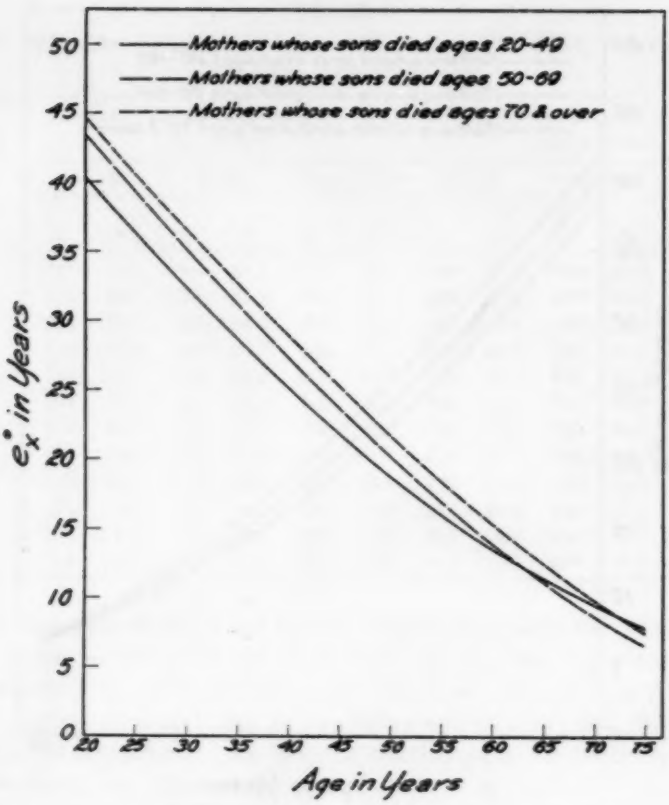


FIG. 9. THE EXPECTATION OF LIFE ($\mathring{e}_x$) IN YEARS FOR MOTHERS WHOSE SONS HAD SHORT, INTERMEDIATE AND LONG DURATION OF LIFE

and 70 and over would be justifiable, and it is impossible to answer the above very adequately on the basis of the data available. But the facts presented in Table 9 may prove suggestive of the general trend of this relationship.

Various combinations of life duration of fathers and mothers are classified into five groups, A-E. In groups A and E, there is only one combination of fathers and mothers and each combination represents the extreme case of short or long duration of life. While it is difficult to differentiate within each of the remaining groups the different combinations of life duration there is a definite difference in life duration

TABLE 9

Changes in Sons' Expectation of Life According to Life Duration of Fathers and Mothers

Group	LIFE DURATION OF PARENTS				SONS' EXPECTATION OF LIFE AT AGES					
	Interval		Mean		20	30	40	50	60	70
	Fathers	Mothers	Fathers	Mothers						
A	20-49	20-49	39.1	38.6	31.5	25.4	20.0	15.2	11.2	7.8
B	20-49 50-69	50-69 20-49	51.6	47.6	32.7	26.5	20.7	15.4	10.7	6.8
	20 & over	20-49	59.3	38.6	32.9	26.9	21.1	15.8	11.1	6.9
	20-49	20 & over	39.1	61.2	33.2	26.6	20.6	15.5	11.1	7.6
C	50-69	50-69	60.4	60.5	33.6	26.7	20.6	15.3	10.9	7.4
	20 & over	50-69	59.3	60.5	34.1	27.5	21.4	16.1	11.5	7.7
	50-69	20 & over	60.4	61.2	34.5	27.7	21.6	16.2	11.6	7.7
	20 & over	20 & over	59.3	61.2	34.9	28.2	22.0	16.5	11.7	7.7
	20-49 70 & over	70 & over 20-49	56.0	61.3	35.4	28.3	21.9	16.4	11.8	8.0
D	50-69 70 & over	70 & over 50-69	66.8	72.1	37.3	29.9	23.3	17.5	12.7	8.8
	70 & over	20 & over	78.3	61.2	37.5	30.3	23.7	17.6	12.4	7.9
	20 & over	70 & over	61.2	78.6	38.0	30.2	23.2	17.1	11.9	7.7
E	70 & over	70 & over	78.3	78.6	39.8	31.8	24.4	17.8	12.1	7.6

of parents between the different groups. This is perhaps best shown by comparing the different combinations of mean life duration of the parents. There is hardly any significant difference between the various combinations in mean life duration within each group, but the demarcation between different groups cannot be mistaken. Now, examining the e_x°'s at each decade of life for the sons of corresponding groups of parents, there is no question as to the trend of the sons' expectation of life with the parents' duration of life. Thus, in spite of the lack of sufficient cases to establish a mathematical relationship between the mean life duration of parents and the expectation of life of their sons, it is clear that the increase in life expectation of the sons runs with a high degree of orderliness according to the change of mean life duration of their parents.

The study of the influence of the complexity of environmental factors upon the duration of life is, of course, beyond the scope of the present paper, but there are some aspects of this question which have a particular bearing on the interpretation of the results already shown and deserve special attention. These will be considered in the following discussion.

It was found in the study of the mortality experience of this family population previously referred to, that the death rate during the period of 1365-1849 had been steadily increasing among young adult and middle-aged persons and their expectation of life, decreasing. In view of this fact and of the small difference of only about thirty years in the period of birth of parents and their offspring, it is likely that parents and their offspring who lived in a period of high mortality would both have shorter length of life while those parents and offspring who lived in a period of low mortality would both have longer duration of life. Is it possible that this similarity of life duration between parents and offspring, presumably due to similar environmental conditions, might account for the marked correlation between parents and sons which has been demonstrated? The answer is negative for the reason that in this family population the maximum change of life expectation at age twenty is about four years in the male and three years in the female and became less than two years in the period of 1700-1849, to which 61 per cent of the fathers, 63 per cent of the mothers and 75 per cent of the sons belonged, while the differences between the groups of sons in respect of their parents are much larger. Moreover, it is impossible on this basis to explain the still greater differences between the groups of

sons when both fathers and mothers dying in the same period of life are considered, because correlation of environmental origin would be the same regardless of the number of factors considered. Nor does it explain the exceedingly high degree of orderliness in the increase of sons' expectation of life with the life duration of their parents.

Next, would short-lived parents have short-lived sons because early loss of parents might have placed the sons in a less favorable environment and consequently shortened their lives? This question cannot affect the results in the present study, since the sons considered here were of age twenty and over. A son who had survived to age twenty had already passed through the stage of life in which he needed parental protection most. It is not likely that his after-lifetime at age twenty and later could be predetermined by his early environmental conditions from which he had successfully emerged.

Finally, it is interesting to note that the maximum change in the expectation of life of this family population which occurred with the change of environment for a period of several hundred years is only about four years in contrast to the largest difference of eight years, which appears when the material is classified according to duration of life of parent and offspring. Of course, it would be unsound from this evidence to argue that heredity plays a more important part than environment in determining the length of life of an individual because it is conceivable that under extremely unfavorable environmental conditions the life duration of individuals of any degree of hereditary superiority could be shortened to such an extent that it would be less than that of persons of poorer heredity placed in more favorable environment, but it is clear that in the case of the people here considered heredity is an outstanding factor in determining whether an individual will be short or long lived.

SUMMARY

In this investigation, the mortality behavior of sons is analyzed in respect of the life duration of their parents, for the purpose of studying the influence of heredity upon the duration of life. The results may be summarized as follows:

1. A son's chance of long life is to a high degree related to the life duration of his parents. The mortality is most favorable for sons whose fathers and mothers both lived to the age period of seventy and over, poorest for sons whose fathers and mothers both died in the age interval

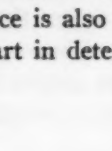
of 20-49 and intermediate for sons whose fathers and mothers both had an intermediate life duration of 50-69. The same finding is true, except that the differences are less marked, when one parent is considered without regard to the other parent, or when the life duration of both parents occurs in the various combinations of age groups possible with the three groups 20-49, 50-69, and 70 and over.

2. There is little difference in the mortality characteristics for sons classified as to their fathers' or their mothers' life duration. In other words, the hereditary influence upon longevity is about the same from the father or the mother.

3. The life curves for parents exhibit very similar differentiation in respect of the life duration of their sons as in the case of sons in respect of their parents' life duration.

4. In view of the limited material, no attempt is made to arrive at a mathematical relationship between the life duration of parents and of their sons, but the trend of increase in the expectation of life for sons at a stated age runs closely parallel to the change of mean life duration of their fathers and mothers, and in a similar manner the life expectation of parents increases with the mean life duration of their sons.

5. Reasons are advanced to show that the marked correlation demonstrated in the present study between the life duration of parents and of their sons cannot be explained on the ground of similar environmental conditions to which both parents and their offspring might be subjected, or of differences in environment of sons of short-lived parents and sons of long-lived parents. Evidence is also brought forward to show that heredity plays an important part in determining the length of life that an individual will attain.



THE RELATION OF THE MODE OF INHERITANCE TO THE SEVERITY OF AN INHERITED DISEASE

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One often encounters the statement in textbooks of medicine that in some instances a disease, such as diabetes, for example, is familial and in other cases it is hereditary. Such a statement is unfortunate, for it at once implies that the cases which are familial, that is, that are found only in the brothers and sisters in a family, but not in the parents or in former generations, are not inherited. They are inherited just as much as are those listed as hereditary, but they exemplify a different type of inheritance. If one wishes to distinguish between those cases that appear suddenly in several of the children of a family without any previous history of a similar disease, and those cases in which parents or former generations in the direct line of descent, or even collaterals, were known to have been similarly affected, one might coin a new word to indicate the latter type of cases. They might be termed *generational* instances, while the term familial was still kept for the former variety. The term hereditary would then be applied with equal correctness to both types.

When one uses the term hereditary, one implies that the character in question has been passed on from parents to offspring via the germ plasm. It does not alter the fact that a character is inherited because it must be derived from both parents before it is manifest in the offspring, while another character, also inherited, comes from only one parent, yet is still visible in the child.

It is the first type of inheritance just mentioned, namely that in which both parents must transmit before the character becomes recognizable as such, that gives us the so-called recessive or "familial" cases. The parents need not, most often *do* not show such a character themselves.

Just as two hidden streams of water, travelling under ground unseen, unknown, suddenly meet, fuse and with the combined strength of the

two, force their way to the surface as a bubbling spring, so two parents may each carry latent a character that is not manifest as such in either of them; but their germ cells meeting, fusing, produce a new individual in whom there springs forth this character in recognizable form. Just as the spring was as truly attributable to one stream as to the other, though neither were visible, so is this new character which apparently springs full grown without any obvious source, derived from both parents and so inherited.

When one studies the inheritance of disease in the human being, one is at once struck with the fact that in man, the same disease may in one family appear to be inherited through generation after generation, without a break; whereas in other families, the same disease may crop up in several children whose parents were normal; or it may appear in only one child as a sporadic example of the disease. It is this peculiarity of the human race, (for in most animals a character is more or less uniform in its mode of inheritance,) that makes the textbooks of medicine state, for example, of diabetes, that cases which are hereditary form about 15 per cent of the whole, that cases which are familial constitute about 7 to 15 per cent, while the sporadic cases make up the remaining part. Because of the erratic modes of inheritance of these diseases in man, all the cases of which cannot be classified in any one category, the tendency is to disregard the study of inheritance in man as being unprofitable.

In reviewing a large series of cases of such diseases, which in some families appear without any former history, and so are familial, and which in other families are generational, several generalizations can be made which appear to me to strengthen materially the idea that such diseases not only are inherited, but follow different modes of inheritance in different families, and that in many instances one cannot say that such and such a disease is transmitted according to any given rule, but must say that in this family it is inherited in one way while in another family it may be transmitted according to entirely different laws.

THE LOCATION OF MUTATIONS

If one conceives that in the course of evolution Nature must have made hundreds of mutations, some of which have gone in the discard, some of which have persisted as variations of the normal as we know it, it is quite comprehensible that the mutation that occurred in the germ

plasm of one person was in such a direction that all the factors necessary for the production of the character, diabetes for instance, were located in one chromosome and if one inherited that, the disease appeared; if not, then one remained normal. In another person, the same mutation appearing suddenly may have affected several chromosomes so that if one got both of them one had diabetes, but if only one of them, then one was apparently normal. If in the latter case, the factors were located in two chromosomes, which were members of a pair, the disease would be inherited as a recessive, whereas if the factors were located in different chromosomes, not mates, it might have a very sporadic type of inheritance, neither dominant nor recessive but partaking of the qualities of first one, then the other in different matings.

If in addition one conceives that this mutation was such that it also determined the severity of the disease, there would be further complications. If a type of the disease, severe because it involved a great deal of the islet tissue of the pancreas, were to arise as a mutation and also have all its factors located in one chromosome, then such a disease would automatically die out. It would appear early in the life of the individual, inasmuch as there would be little normal tissue left to function and the person would die before reaching the age of maturity, and so before he could pass on this newly appearing mutation that was evident in him for the first time. It would be a mutation that would rise above the surface of the Sea of Being to be submerged again before it would wreck the life of more than the few children in that family in whom it might chance to appear.

But suppose that it was one which, although having the capacity to kill early due to the severity of the disease, was also one which appeared in the chromosomes that were mates or allelomorphs. It then would be of such a nature that it could appear in the offspring of parents that were themselves not affected. Under such circumstances, the patient would die early of the disease, but that would not in the least affect its reappearance in other children of the same family nor in subsequent generations, for some of the children would receive, not two factors, but merely one for the disease. They would then be like their parents, free from it, but capable of having affected offspring if they mated with a person like themselves who carried one factor latent for the production of this particular character. Thus there would be no direct inheritance, but the collaterals in the same or different generations might show the condition. The affected person would die young as in

the other case, but the stream of inheritance would flow on through others not affected.

Is there any evidence apart from the actual pedigrees, to support the idea that the same disease might be inherited in several different ways, or might one claim that rather elaborate and complicated laws of transmission governed all the cases and that were these laws known, all the pedigrees would be seen to express them perfectly? If the supposition that the disease arose in some families as a dominant and in some as a recessive, and that there were also factors linked with this that governed the severity of the disease, and hence the age at which it appeared, then it would be only in those families in which the disease was not severe that the dominant type would be propagated. If it were being inherited as a recessive, not only the mild variety, in which the person was not affected until some time past the age at which he could reproduce, but also the severe form in which he died in childhood would reappear erratically through the generations. Hence studying the age of onset of the disease in different families would be of assistance in determining whether all cases were being inherited according to the same laws.

The average age of onset of any disease which showed variations in its mode of inheritance should be uniformly higher for those families which showed the generational type of inheritance, than for those which showed the familial type; inasmuch as the former would include merely the mild cases; the latter would include the severe as well as the mild. It would explain too why the textbooks state that in the majority of cases, if the disease starts at a young age it is far more severe than if it begins later in life. Such predilection for youth is not due to the greater susceptibility of the child to these maladies, but to the mode of inheritance, whereby most of the cases in adults have been restricted to the mild variety.

VARIATION IN AGE AT ONSET WITH TYPE OF INHERITANCE

Let us examine the data to see if any support is lent to this theoretical consideration. Consider first the disease of diabetes mellitus, which we have been using as an example through the discussion. Collecting from the literature cases in which the disease was familial, and so apparently due to recessive inheritance, or even generational, but not direct in its mode of transmission, I obtained fifty-nine cases in which the age of onset was given. The average was 11.2 years. This was well below the age of reproduction. There were sixty-three cases

in which the inheritance was generational and direct, so that the disease may with a fair amount of justice be considered to have arisen through a mutation acting as a dominant character. The average age of onset in these cases was 30 years; an age well above that at which the individual could have reproduced his defect.

The data on age of death would have shown the same thing, namely that the age of death from the disease was in the first class of cases below the age at which the individual would have reproduced, while in the second case it naturally would have been beyond that. So far then the actual observations bear out the theoretical deductions, namely, that diabetes is not inherited according to some more or less complex scheme which if understood would explain all the cases which are not obviously due to infection or new growths of the pancreas, etc., but which depend upon an inherent defect of the islet tissue. The comparison of the ages of onset shows that the disease in its acute form which attacks the young is inherited as a recessive, while in its mild form it may be inherited as a dominant or recessive.

If such an explanation is true, it should apply then to other diseases which do not have a uniform mode of inheritance, and which are either fatal to the patient directly, or so transform him that he no longer is to be regarded as a potential parent, his physical attributes being such that in the selection of mates he is left aside. We should find that in the familial, indirect type of inheritance the age of onset would be younger than in the cases in which the transmission is in the direct line.

Let us consider some of the diseases of the nervous system, which are examples, *par excellence*, of variation in their mode of transmission in different families. Take the progressive muscular atrophy of the adult and of the infant, the Werdnig-Hoffmann type. What do we find here? The average age of onset of twenty-five cases of the Werdnig-Hoffmann atrophy of children was at 9 months, the average age of death at 2.4 years. Obviously the parents had to be normal in these cases, none of the affected ever surviving infancy; so that the disease was inherited as a recessive. If ever a mutation occurred in the germ plasm of a person whereby this disease could be inherited as a dominant, it would be wiped out at once with the death of the tiny sufferer. Of course one might consider that these cases, or at least some of them, were just that type, namely dominant mutations which were discarded as soon as tried.

The adult form of progressive muscular atrophy was found in twenty-

six cases in which the line of descent was direct, and so probably of the dominant variety, and in which the age of onset was given. This average was 43 years. Again the dominant form is found to be the milder or at least that attacking later in life.

In hereditary spastic paraplegia the two modes of inheritance are quite common. There were found in the literature 120 cases in which the disease was familial, and in which the age of onset was given. The average was 9 years. The range was from 9 months to 71 years, thus showing that there are recessive mild cases as well as dominant mild cases; but with the average at 9 years, it also shows that the majority of cases were in very young children. There were eighty-one cases in which the line of descent was direct and in these the average age of onset was 17.8 with a range from 4 to 50 years.

The same thing is true of peroneal atrophy. The recessive or familial cases in which the age of onset was given numbered thirty, and the average age was 10.5; while in the seventy-six cases in which the disease behaved as a dominant the average was 16. In muscular dystrophy there were fifty-seven familial cases with an average of 9.3 years for the age of onset, while there were twenty-four cases which showed direct inheritance and which had an average age of onset at 15.6. In Friedreich's ataxia, the familial cases were 139 in number with an average age of onset at 10.5 years, while the ninety-six cases in which the descent was direct showed an average of 16. The two corresponding figures for the eighteen familial and twenty-eight generational cases of cerebellar ataxia were 17 and 24 years.

Going outside the realm of the nervous system and viewing the cases of angioneurotic edema, we find six cases in which the disease was familial in type, the parents being normal. All these children were in the same family and all succumbed a few weeks after birth, the average duration of life being 7½ weeks. In cases showing the dominant mode of inheritance it is stated that the onset is usually between 15 and 20 years. I found ten cases in which the actual age at which the disease began was listed. The average was 15.4 years.

Polycystic disease of the kidneys is a condition that kills early or late according to the amount of tissue involved, and in that respect is like diabetes. One might get along well for years with a partial involvement, but not be able to survive with complete destruction of the secreting portion of the kidney by cysts. There are numerous reports in which the disease affects either one child in the family only, or in

which more than one are affected, and in which the lesion is so extensive that the child not only cannot live after it is born but in which it cannot even be born alive. The abdomen is so distended that normal birth is impossible and the child has to be extracted piecemeal. This type of inheritance might be regarded as recessive in character, or as a dominant mutation which appears in the germ plasm of the parents, only to be snuffed out of existence immediately. In the dominant mode of transmission, there were forty-three cases with an average age of onset at 31.7 years.

Hereditary albuminuria shows somewhat the same relations, but inasmuch as it is not so necessarily fatal, even the dominant mode of transmission shows a relatively young age of onset. There were eight cases listed in the familial class with an average age of onset at 4 years. The dominant cases were nine in number with an average of 8.1 years.

Thus a review of widely different diseases which show an apparently varying mode of transmission in different families leads to the same conclusion in all, namely that the families in which the disease appears without any previous history of it, or in which the affected persons are always in collateral branches, never in the direct line of descent, show a much earlier age of onset and a more severe type of disease than do the families in which the line of descent is direct. Thus there is a rational basis for the observation that a disease (apart from the infectious diseases of childhood) which appears in either young or old, is far more apt to be of the severe variety when found in young children. The rational basis is the mode of inheritance which the disease shows in that family, and depends upon the way in which the mutation causing the departure from normal first affected the chromosomes in the ancestors of the patients investigated.

Just what name should be given to this observed generalization is hard to determine. One hesitates to call it a law, but it appears to be a relationship that runs through quite a number of inherited diseases. Its correlary is one which I have never seen stated, perhaps because so obvious, namely that if a disease or abnormality is inherited as a dominant, it is one which is not fatal, or if fatal, it is one which commences after the period of adult life is reached. The diseases which are fatal soon after their appearance and which are apt to appear in early childhood are always recessive in character, necessarily so.

CONSTANCY OF AGE OF ONSET WITHIN A FAMILY

There is another fact which I should like to emphasize, that is connected with the one just discussed, and it is an added proof to the conclusions just drawn. One finds in discussing any family in which an hereditary disease is found, that on the whole the age of onset in the various members of that family is about constant. It may differ widely from the age of onset of the next family found with the same condition, so that the range of ages in many families may run the gamut of the entire span of life, but the children in one family will all be affected at approximately the same age. That strengthens the conception that not only is there inherited the disease process itself, but also its extent or its severity, which in turn determines how long the person affected will be able to live before showing the degenerative process.

For example, in hereditary spastic paraplegia the age of onset for the familial cases was stated to be 9 years. Consider the individual families, however; the ages of onset for two children were 6 and 6; while another family showed the ages of 27, 27 and 27. Still another family had its three children affected at 6 months, 6 months and 20 months. In another the three were affected at 2 years of age, while in another they were 9 and 12; in a third family, 3½ and 5. In one family there were eight children all affected between 12 and 18 months. In another the ages were 15 and 17 years. In the dominant mode of inheritance the same rule holds; one family of children was affected at 30, 31, 35, 40 and 52.

Consider muscular dystrophy, for example. In one family, the onset was at 4 and 6 years; in another at 1 year in the two boys; in another at 19 in the two cases; in still another at 1½, 5 and 5; in another at 9 and 9; or at 14 and 15. In a very interesting family in which all thirteen patients were derived from the same two unrelated men some eight generations previously, the onset in these persons, who occurred in ten families, was at 2, 7, 5, (these last two being sisters); 15, 15, 8, 10, 12. In only two instances did the disease commence in adult life, in one case at 40, and in a second one at 24.

In Friedreich's ataxia, the same thing is noted. In one family, the age of onset was at 5½, 6½, 6½; in a second at 12, 12 and 10; or at 18 and 19; or at 18, 20, 26, 18 and 17; a group that is much later than the first group reported; or at 18 and 21; at 2 and 3; at 17 and 19. So we might go on, indefinitely, showing that although the range is fairly wide in many of these diseases, the age range in any one family is as a

rule well limited. This must mean then that there is a specific factor inherited which regulates the amount of tissue that is involved in the disease process and the length of the life of the nerve cells and nerve fibres before they begin to show their atrophic condition. This we might term a virulence factor.

Still another generalization may be drawn from these cases of inherited diseases. Not all the symptoms that are possible in any one disease necessarily appear in any one individual, and it is of interest to find that if one member, in a family affected with Friedreich's ataxia for instance, complains of pains in the legs, most of the others will have the same thing; or if scoliosis be the first defect noted in a child, the subsequent members of the family to develop the disease will frequently show scoliosis before they do any other trouble. If nystagmus is present in one child, then nystagmus is apt to be present in the rest of the family who are affected, allowing time to elapse of course for the development of the various symptoms. Thus not only is the time or virulence factor fairly constant for a family, but so is the extent factor. The groups of nerve cells, the tracts of fibres that are attacked, and the sequence in which they become involved are apparently more or less the same in the children in the same family. In one family suffering with diabetes, six persons had had a leg amputated for gangrene, yet many diabetics go through life with no sign of gangrene.

THE LAW OF ANTICIPATION

A generalization noted in the study of inherited diseases was made years ago, nor am I able to say just who was responsible for it, although I myself first found it referred to in the writings of that great student of human inheritance in eye defects, Edward Nettleship. He designated it as the law of anticipation, meaning that a disease which was inherited in the direct line of descent tended to appear at an earlier age in the children than it did in the parents. For example, in progressive muscular atrophy, the age of onset in six cases in the first generation was 51 years. For ten cases in the second generation, it was 43.4; for seven cases in the third it was 43; for two cases in the fourth it was 31.5; and for the one case in the fifth it was 13. The same decrease is noted in muscular dystrophy, the average for the first being 27, for the second 14, for the third 12. When we look at the cases of polycystic kidney, we find the same thing. There were eleven cases reported which were listed as the first generation, with an average age of onset at 47. There

were twenty-one cases in the second generation with an average of 29. In the third generation the average of eight cases was 25, while in the fourth the average of two cases was 14.5.

It would be assumed then that one need not worry about these diseases, that they tended to wipe themselves out by occurring at progressively earlier ages. This unfortunately is not true. If it were, we would have dispensed with these inherited defects long ere this, but we find them constantly increasing, partly because surgical skill has tended to correct those which can be modified, and modern philanthropy protects and cares for those who cannot be rehabilitated. How then reconcile the law of anticipation and the increasing amount of inherited disease? Because, although the *average* age of onset is lower, there are some among the affected children in whom it shows at a time as late or later than it did in their parents. For example, a mother who had peroneal atrophy, developed her disease at 20. Two of her three boys who became affected showed their trouble at 12, but the third son did not come into his heritage until he was 35. Thus the average for that generation was a little below the time at which the mother was affected, but one person in it became diseased so late that he started, as it were, a new cycle, thus allowing the anticipation tendency to evince itself again in his offspring.

Davenport in referring to the tendency for Huntington's chorea to appear earlier in the offspring than it did in the parents, states that this is more apparent than real. The grandparents are a selected class, so he says, selected on the basis of the late onset, otherwise they would not have become grandparents. The ones who were affected early in that generation would never get to be grandfathers and hence the age of onset for the first generation would appear to be unduly high. Such an argument is very sound in some respects, but loses its force when we leave out of consideration in any given pedigree, the one progenitor who stands as the sole representative of the first generation and consider all his children, whether they ever become parents or not, and the ages of their onset, and all the children of the third generation, etc., with their ages of onset, and determine whether the age for *all* the third generation is less than the age for *all* the second. When we do this we then overcome Davenport's objections, for we consider not only those of the second generation who lived to marry but also those who did not; thus eliminating the statement that we deal in the first generation with only a selected class. When we make this allowance we still have

the average age for all in the third generation less than the average age for all in the second; and the average age for the entire fourth generation which is affected, less than the average for all those of the third, regardless of whether they become parents or not. The law of anticipation seems to be a real fact in hereditary diseases.

SUMMARY

From a survey of published cases of inherited diseases, in which the type of inheritance varies in different families, several generalizations may be made. The first is that some diseases are inherited in different families according to different laws. The families in which occurs the dominant mode of transmission of a disease which is of such nature that it is a menace to the life of the individual are usually affected either with a milder form of the disease or are affected later in life than are those families in which the disease appears without any previous history of it. Although the recessive mode of inheritance as well as the dominant may be found in the mild form, or that which appears later in life, as a rule, it is the former only which characterizes those cases which are severe and early in their onset. It is the mode of inheritance of these diseases which establishes the relation between the severity of the disease and the youth of the patient.

The second is that there is also inherited a factor which one might term the time or virulence factor, which makes the disease appear at relatively the same age in children of the same family.

The third is that in diseases of the nervous system, the sequence in which symptoms appear, is regulated by the order of the involvement of the nerve tissue, and there is a tendency for all children of the family to show the same symptoms, and in the same order. This merely means that the disease process proceeds in the same way in the children of the same family.

The fourth is, that although the law of anticipation is exemplified in many of the diseases which are inherited in the direct line, a disease does not thereby tend to eradicate itself, there usually being several members of a generation in whom the disease starts sufficiently near the age at which the parent was affected or even beyond that, to ensure his being the starting point of a new cycle.

STUDIES ON HUMAN LONGEVITY
V. CONSTITUTIONAL FACTORS IN MORTALITY AT ADVANCED AGES[1]

BY RAYMOND PEARL AND T. RAENKHAM

I

As a part of a comprehensive study of longevity, senescence, and senility, it is necessary to make a thorough and critical examination of the causes of death of persons attaining extremely advanced ages. Specifically, for the purposes of the present discussion, the primary problem here may be formulated in the following way:

From what diseases or causes do persons die whose deaths occur at ages of 90 years and above?

This primary problem at once suggests a number of correlative questions, among which two in particular may be mentioned.

Are the relative frequencies of the different causes of death among those dying at ages of 90 years and over similar to, or different from, what they are among those dying in other periods of life, particularly those dying as young adults, or as middle-aged persons not yet obviously senescent?

What organ systems are most resistant to the wear and tear of living?

This last question implies clearly the interest of the problem from the standpoint of constitution theory. The idea that the different organ systems of the same individual may have quite different biological worth (*i. e.*, ability to resist the stresses and strains of functioning, of infection, of trauma, etc.) is a relatively old one.[2] In a series of contributions

[1] From the Department of Biology of the School of Hygiene and Public Health, Johns Hopkins University.

[2] *Cf.*, Adler, A. Studie über Minderwertigkeit von Organen. *München* (J. F. Bergmann), 1927. Pp. 92. (This is a revised reprint. The book was first published in 1907.) Adler was the first to develop this idea systematically in its relation to human pathology. He rightly says (p. 4): "Die Lehre von der Minderwertigkeit der Organe Probleme in Angriff nimmt, die zu den wichtigsten der

during the past ten years Pearl[3] has developed the idea that an index of the relative biological worth of various organ systems may be obtained by examining the proportionate mortality assignable to each of them. This suggestion has been adopted by other workers[4] in connection with their special problems.

When dealing with the mortality at ages of 90 and above this constitutional aspect takes the foremost position. Persons who survive to such advanced ages give evidence, by the fact itself, of unusual powers of resistance to disease, which is to say, of exceptional soundness of constitution. The quantitative degree of exposure-to-risk of falling ill is primarily proportional to the length of time the individual lives. When a person has lived to the age of 90 or above he has had an extremely great exposure to risk. But his powers of resistance have been such that, even though he has had diseases, he has been able to survive them. Yet ultimately even such persons succumb. And in the great majority of cases they succumb because some particular organ system at last breaks down, no longer able to withstand the demands made upon it to carry on its part in the integrated whole which is the living individual. The other organs and tissues of the body may still be quite capable of carrying on indefinitely, and in many cases are, if one may judge by what is found at autopsy.[5] In the causes of death of such people there

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Pathologie gehören." But in more general terms the idea of differing biological worth or fitness of organ systems was implicit in Roux's discussion of the "Kampf der Teile."

[3] Pearl, R. Certain evolutionary aspects of human mortality rates. *Amer. Nat.*, Vol. 54, pp. 5-44, 1920; Studies in Human Biology, *Baltimore* (Williams and Wilkins), 1924, Chap. VII; Evolution and mortality, *Quart. Rev. Biol.*, Vol. 3, pp. 271-280, 1928; Biological factors in negro mortality, HUMAN BIOL., Vol. 1, pp. 229-249, 1929. Other papers have been published on the same topic, but need not be cited here. References to them will be found in the works here cited.

[4] *Cf.*, for example, Schwarz, N., Ein Versuch der biologischen Klassifikation der Krankheiten nach der ärztlichen Untersuchung der Jünglinge, *Zeitschr. f. Konstitutionslehre*, Bd. 12, pp. 779-789, 1926, wherein Pearl's classification is applied to the analysis of the morbidity of schoolchildren. Also see Fox, H., Some observations on comparative constitution in man and the lower animals, *Proc. Amer. Phil. Soc.*, Vol. 68, pp. 27-51, 1929. In this paper a similar classification is applied to the autopsy records of a large zoological garden. Tandy, E. C., Secular changes in mortality rates connected with certain organ systems, HUMAN BIOL., Vol. 3, pp. 494-528, 1931, has applied the Pearl classification in a study of mortality trends and correlations in different populations.

[5] *Cf.*, for example, autopsy records of persons who were alleged to be centenarians at the time of death, and who were certainly of extremely advanced age,

is seen reflected with considerable clearness the *locus* of ultimate weakness in the bodily constitution.

II

The biology and diseases of senility have been discussed from the remotest antiquity. Hippocrates (*Aphorisms*, Sect. 3, Aph. 31) includes a good part of all that has been observed and said by later writers on the subject in the following terse statement: "To old people dyspnoea, catarrhs accompanied with coughs, dysuria, pains of joints, nephritis, vertigo, apoplexy, cachexia, pruritus of the whole body, insomnolency, defluxions of the bowels, of the eyes, and of the nose, dimness of sight, cataract (glaucoma), and dullness of hearing." [Adams's translation.]

Galen (*Hyg.*, v) put what he regarded as the physiological essence of old age in the words: "Old age is cold and dry." Therefore he proposed to treat it with diluents and calefacients, such as hot baths of sweet waters, drinking wine, and taking food that is moistening and calefacient. His directions for the regimen of the aged were widely copied by a succession of early medical writers, as Oribasius (*Synops.*, v. 18), Aëtius (iv, 30), Paulus Aegineta (*Bk.* I, *Sect.* xxiv), the Arabians, Alsaharavius (*Pract.*, xxvii, 10), Avicenna (*Cantica*, i, 3), and Averhoes (*Comment.*, tr. i).[6]

The "coldness" and dryness of the senile has been apparent to observers throughout the ages. Burton, writing in the middle of the seventeenth century, in the *Anatomy of Melancholy* (*Partition* 1, *Section* 2, *Member* 1, *Subsection* 5), says:[7]

"The first of these, which is natural to all, and which no man living can avoid, is old age, which being cold and dry, and of the same quality as melancholy is, must needs cause it, by diminution of spirits and substance, and increasing of adust humours; therefore Melancthon avers out of Aristotle, as an undoubted truth, *Senes plerunque delirâsse in senectâ*, that old men familiarly dote, *ob atram bilem*,

such as Harvey's autopsy of Old Parr (The Works of William Harvey, *London* (Sydenham Soc.), 1847, pp. 589-592; reprinted in HUMAN BIOLOGY, Vol. 3, pp. 138-142, 1931) and Rolleston's autopsy of John Pratt (Scientific Papers and Addresses by George Rolleston, *Oxford* (Clarendon Press), 1884, Vol. I, Chap. XII). It has been said that persons dying at 60 often show at autopsy more of the signs which are supposed to characterize extreme senility than do centenarians.

[6] This paragraph is based upon Francis Adams's translation of and commentary upon "The Seven Books of Paulus Aegineta." *London* (Sydenham Society), 1844. Vol. I, pp. 36 and 37.

[7] The quotation is made from the "new edition" published by Chatto and Windus, London, 1898, p. 136.

for black choler, which is then superabundant in them: and Rhasis, that Arabian physician, in his *Cont. lib. 1, cap. 9,* calls it 'a necessary and inseparable accident,' to all old and decrepit persons."

Charcot [8] in his historical account of early medical writings on old age refers to only two treatises of any importance in the eighteenth century, Floyer's "Medicina Geronomica," 1724; and Fisher's "Tractatus de senio," 1766. Others might be mentioned, but as Charcot quite truly says (*loc. cit.*, p. 18): "Most of the medical works of the past century which touch in a special manner, upon the senile period of life, have a literary or a philosophical bearing; they are more or less ingenious paraphrases of the famous treatise 'De Senectute' of the Roman orator." [Marcus Tullius Cicero.]

Early in the nineteenth century Brossard-Ysabeau [9] discussed the diseases of old age with considerable acumen, but without any statistical data or conceptions. He regarded senility as characterized pathologically chiefly by two things: an "affaiblissement général" and a steady, increasing accumulation of calcium phosphate in the body. Specifically he singles out as especially important diseases of old age three (cataract, hardening of the tympanic membrane with obstruction of the Eustachian tube, and gout) as due to the general desiccation accompanying age. These are, of course, singularly non-lethal affections. As more important he regarded diseases due to obstruction of the blood vessels, mentioning first what we should now call arteriosclerosis, then tumors, kidney stones, paralysis, dropsy and mucous catarrhs, particularly of the lungs, the intestines, and the bladder. In general the diseases of old age are clinically "adynamique," and may, in fact, well be designated as *asthenic.*

Brossard-Ysabeau's thesis appeared in the same year (1815) as the "Traité de Médecine Clinique" of Pinel, who was physician to the Salpêtrière. Pinel pointed out the desirability, without doing much of anything else about it, of special discussion and treatment of the diseases of senility.

During the nineteenth century a considerable number of more or less valuable treatises on this subject have appeared, notably those of

[8] Charcot, J. M. Clinical Lectures on the Diseases of Old Age. Translated by Leigh M. Hunt. With Additional Lectures by Alfred L. Loomis. *New York* (William Wood and Co.), 1881. Pp. xv + 280.

[9] Brossard-Ysabeau, L. Considérations sur les Caractères de la Vieillesse, et sur les Affectations qui sont propres à cet âge; Thèse inaugurale. *Paris* (Imp. de Didot Jeune), 1815. Pp. i-vi + 7-28.

van Oven,[10] who singles out for discussion as the specially important diseases of old age, gout, cancer, diseases of the digestive organs, of the urinary organs, of the "sanguineous system," of the heart and arteries, apoplexy and hemiplegia, diseases of the skin, and *tussis senilis*; Canstatt[11] and Durand-Fardel,[12] whose works are comprehensive clinical treatises now rather out of date, but authoritative in their time.

At the present time the outstanding clinical pathological treatise on old age is Schlesinger's,[13] which was published as the eighth supplementary volume to Nothnagel's "Spezielle Pathologie und Therapie." It is a thorough and comprehensive piece of work. Other recent works of the same character which should be mentioned are those of Boy-Teissier,[14] Rauzier,[15] Pic and Bonnamour,[16] Thewlis,[17] and Nascher.[18] Three more general treatises on old age, which contain interesting discussions of the diseases of this period of life are those of Hall,[19] Vidal,[20] and Warthin.[21]

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[10] van Oven, B. On the Decline of Life in Health and Disease, Being an Attempt to Investigate the Causes of Longevity; and the best Means of Attaining a Healthful Old Age. *London* (John Churchill), 1853. Pp. xx + 300.

[11] Canstatt. Die Krankheiten des höheren Alters und ihre Heilung. 2 vol. Erlangen, 1839.

[12] Durand-Fardel, M. Traité pratique des maladies des vieillards. Deuxième Edition. Paris (Germer Baillière), 1873. Pp. lv + 815.

[13] Schlesinger, H. Die Krankheiten des höheren Lebensalters. 2 vol. *Wien und Leipzig* (Alfred Hölder), Vol. I, 1914, pp. viii + 611; Vol. II, 1915, pp. 541.

[14] Boy-Teissier. Leçons sur les maladies des vieillards, faites à l'Ecole de médecine de Marseille. De la senilité en général. *Paris* (O. Doin), 1895. Pp. 296.

[15] Rauzier, G. Traité des maladies des vieillards, préface de M. le professeur Grasset. *Paris* (J. B. Baillière), 1909. Pp. 692.

[16] Pic, A., and S. Bonnamour. Précis des maladies des vieillards, préface du Prof. Bouchard. *Paris* (Doin), 1912. Pp. vi + 890.

[17] Thewlis, M. W. Geriatrics. A Treatise on the Prevention and Treatment of Diseases of Old Age and the Care of the Aged. 2nd Edit. *St. Louis* (Mosby), 1924. Pp. 401.

[18] Nascher, I. L. Geriatrics; the Diseases of Old Age and Their Treatment, including Physiological Old Age. Home and Institutional Care, and Medico-legal Relations. With an Introduction by A. Jacobi. 2nd Edit. *Philadelphia* (Blakiston), 1916. Pp. xx + 527.

[19] Hall, G. Stanley. Senescence. The Last Half of Life. *New York and London* (Appleton), 1922. Pp. xxvii + 518.

[20] Vidal, C. Le vieillard. Quelques considerations. *Paris* (Blond et Gay), 1925. Pp. 540.

[21] Warthin, A. S. Old Age the Major Involution. The Physiology and Pathology of the Aging Process. *New York* (Hoeber), 1930. Pp. xvi + 199.

While the works which have been cited above treat the subject of the pathology of old age from a variety of viewpoints, unfortunately that of modern medical statistics (biostatistics) is, generally speaking, not one of them. The diseases of the aged are discussed rather from the clinical point of view, primarily with an eye to their treatment, rather than from the point of view of their relative or comparative fatality. It is probably fair to say that nearly or quite as much space, perhaps more, has been devoted to gout and rheumatism in the older medical literature of senility as has been given to diseases of the heart or to cerebral hemorrhage. Yet gout, while doubtless an exceedingly painful ailment, is quite insignificant as a cause of death.

There have, however, been a few studies of the problem with which the present paper has to do. In 1885 G. M. Humphry [22] published the results of a British Medical Association collective study of 500 people over 80 years of age, in which particular attention was devoted to constitutional questions. His general position was that (p. 928):

"The first requisite for longevity must clearly be an inherent or inborn quality of endurance, of steady persistent nutritive force, which includes reparative force and resistance to disturbing agencies, and a good proportion or balance between the several organs. Each organ must be sound in itself, and its strength must have a due relation to the strength of the other organs. If the heart and digestive system be disproportionately strong, they will overload and oppress the other organs, one of which will soon give way; and, as the strength of the human body, like that of a chain, is to be measured by its weakest link, one disproportionately feeble organ endangers or destroys the whole. The second requisite is freedom from exposure to the various casualties, indiscretions, and other causes of disease to which illness and early death are so much due. Now, in both these—notably in the second—woman has the advantage over man, and she consequently attains to greater age. In the report of the Registrar-General for 1873, eighty-nine persons were returned as dying at or over the age of 100. Of these, ten only were males; and the superiority of female life is well known by insurance-officers to exist, notwithstanding the higher rate of mortality that has been observed during the child-bearing period, and which, there is reason to think, is now slowly disappearing. That this superiority is not entirely due to the comparative freedom from exposures and to the greater temperance in the woman, but is partly a result of a stronger or more enduring inherent vitality, is shown by the fact that, even in the first year of life, when the conditions and exposures of male and female infants are the same, the mortality of girls is less than that of boys. A somewhat larger number of boys are born, but they are more difficult to rear; so that the females soon gain the numerical lead, and maintain it, with almost steadily increasing ratio, to the end."

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[22] Humphry, G. M. Old age and the changes incident to it. *Brit. Med. Jour.*, Vol. I for 1885, pp. 927-931, May 9, 1885.

Humphry found that both males and females of average weight and stature lived longer than those much above or below average. The average number of teeth in the men was 6, and in the women 3. Of 221 men 57 were edentulous, and of 234 women 113 were in that condition. Humphry's findings regarding arteriosclerosis are extremely interesting. He says (p. 929):

"It may, I think, in like manner, be said, with regard to the calcification of the arteries, that it is the result of a morbid process intruding itself upon, interfering with, and arresting, the normal progress of senile development. That it is not, at any rate to a perceptible amount, a usual accompaniment of old age, is shown by the fact that, in 362 returns respecting the condition of the arteries in persons over 80, these vessels are stated to be knotty in only 40, and even in 257; they are noted as being tortuous in 71. Moreover, the pulse is reported as compressible in 311, and incompressible in 72, in the returns relating to it. In the great majority of cases, therefore, the arterial system appears to present a healthy condition in those who attain to great age."

Friedman [23] puts desiccation as the primary physiological cause of senility. He says that the aged are relatively immune from eruptive fevers, typhoid, pulmonary tuberculosis, perhaps because old tissues are not good media for such diseases, but he maintains that the old are especially liable to pneumonia and erysipelas.

Thompson [24] discussed the mortality records of 1910 in the U. S. Registration Area. Unfortunately he made no distinction as to sex or color, but lumped all deaths together. This means that the considerable errors inherent in records of mortality among the colored at these advanced ages, which are discussed in detail in the next section of this paper, make it impossible to form any reliable judgment about the mortality relations among the whites from his discussion.

Thompson lists the more important causes of death in descending order as Diseases of the circulatory system [this is the International List class]; Diseases of the respiratory system [Int. List]; Diseases of the digestive system [Int. List]; Bright's disease; Apoplexy; Influenza; and Cancer. He is of the opinion that (p. 280): "Heredity appears to be a much stronger factor in producing nonagenarians than any special habits, as of diet or hygiene, for the latter are found to present the greatest variations in different cases—hence extreme longevity is largely a matter of natural selection."

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[23] Friedman, H. M. Senility, premature senility, and longevity. *New York Med. Jour.*, Vol. 102, pp. 65-71, July 10, 1915.

[24] Thompson, W. G. Centenarians and nonagenarians. *Med. Rec.*, Vol. 83, pp. 277-280, Feb. 15, 1913.

III

In the five years 1923-1927 inclusive there occurred in the U. S. Registration Area [25] a total of 5,985,833 deaths, of which 2,811,195 were deaths of white males; 2,394,231 deaths of white females; 409,064 deaths of colored males; and 371,343 deaths of colored females. Out of this total of nearly six million deaths, 85,039 were recorded as of persons 90 or more years of age at the time of their demise. These were distributed by race and sex as follows: 29,317 white male deaths at ages of 90 and over; 43,003 white female deaths at ages of 90 and over; 5,359 colored male deaths at ages of 90 and over; and 7,360 colored female deaths in the same age category. It is these deaths of persons 90 and over years of age that we shall proceed to discuss.

The magnitude of the material is statistically respectable. The registration of deaths in the United States during the years specified

TABLE 1

Total Deaths at Ages 90 Years and Above, in the U. S. Registration States, 1923-1927 Inclusive, Classified by Organ Systems

	ORGANOLOGICAL CLASSES	MALES		FEMALES	
		White	Colored	White	Colored
	All Causes	29,317	5,359	43,003	7,360
Class	I. Circulatory system, blood	10,374	1,068	14,576	1,469
	II. Respiratory system	4,130	476	6,536	746
	III. Primary and secondary sex organs	550	86	485	75
	IV. Kidneys and related excretory organs	3,421	734	3,733	686
	V. Skeletal and muscular systems	50	30	124	38
	VI. Alimentary tract and associated organs	1,423	251	2,105	320
	VII. Nervous system and sense organs	3,438	477	5,440	777
	VIII. Skin	344	13	452	16
	IX. Endocrine system	91	18	143	19
	X. All other causes	5,496	2,206	9,409	3,214

[25] These statistics and those which follow in the tables of this section of the present paper are derived from the official annual volumes of *Mortality Statistics* published by the Bureau of the Census of the U. S. A., for the years 1923-1927 inclusive.

was well organized and working smoothly, and the records may be looked upon as quite as accurate as any death records which have ever been made in this country. Furthermore there was no particular disturbance, epidemic or otherwise, of the smooth and regular flow of mortality during these five years 1923-1927.

In Tables 1-11 inclusive these deaths of aged persons (90 years and over) are classified according to Pearl's (*loc. cit.*) biological classification of mortality. Table 1 gives the absolute number of deaths assignable to each of the organ system classes. Tables 2-11 inclusive give, first, the detailed causes of death assigned to each organ system class; and, second, the percentage of the total mortality contributed by each cause and each biological class of causes.

TABLE 2

Percentage of Deaths at Ages 90 Years and Over Assignable to the Circulatory System, Blood, Blood-forming, and Blood-storing Organs. Class I

INTERNAT. LIST NO.	CAUSE OF DEATH	PER CENT OF TOTAL DEATHS			
		Males		Females	
		White	Colored	White	Colored
90	Other diseases of the heart	22.544	14.367	22.605	14.062
91b, c	Other diseases of the arteries	10.519	4.087	9.448	4.185
74a	Cerebral hemorrhage	9.845	6.101	10.613	7.662
151	Gangrene	0.846	0.467	0.500	0.557
89	Angina pectoris	0.658	0.224	0.533	0.217
74b	Cerebral embolism and thrombosis	0.348	0	0.484	0.190
88	Endocarditis	0.298	0.075	0.288	0.121
83	Softening of the brain	0.153	0.075	0.183	0.082
92	Embolism and thrombosis	0.130	0.019	0.179	0.068
58	Anemia, chlorosis	0.116	0.056	0.105	0.054
87	Pericarditis	0.078	0.093	0.051	0.068
91a	Aneurysm	0.051	0	0.028	0.027
41	Purulent infection, septicemia	0.044	0.075	0.044	0.082
5	Malaria	0.034	0.410	0.035	0.448
93	Diseases of the veins	0.031	0.019	0.033	0.014
95	Hemorrhage without specified cause	0.014	0	0.014	0.014
96	Other diseases of the circulatory system	0.010	0.037	0.012	0.041
65a	Leukemia	0.007	0	0.005	0
65b	Hodgkin's disease	0.003	0	0.005	0
94	Diseases of lymphatic system	0.003	0	0.005	0
7	Measles	0	0.019	0.007	0
64	Diseases of the spleen	0	0	0.005	0
	Totals	45.732	26.124	45.182	27.893

TABLE 3

Percentage of Deaths at Ages 90 Years and Over Assignable to the Respiratory System. Class II

INTERNAT. LIST NO.	CAUSE OF DEATH	PER CENT OF TOTAL DEATHS			
		Males		Females	
		White	Colored	White	Colored
100a	Bronchopneumonia	3.998	1.361	4.692	1.630
101a	Lobar pneumonia	2.609	2.109	2.484	1.807
11a	Influenza (with pulmonary complications)	1.146	0.933	1.307	1.454
99a	Acute bronchitis	0.805	0.149	0.921	0.190
103	Congestion and hemorrhagic infarct of the lung	0.880	0.187	0.932	0.190
99b	Chronic bronchitis	0.771	0.261	0.712	0.231
99c, d	Bronchitis (unspecified)	0.583	0.243	0.833	0.324
101b	Pneumonia (unspecified)	0.566	0.784	0.549	0.774
105	Asthma	0.174	0.224	0.172	0.231
31	Tuberculosis of the respiratory system	0.136	0.467	0.172	0.367
100b	Capillary bronchitis	0.113	0.056	0.107	0.014
102	Pleurisy	0.075	0.112	0.072	0.082
107	Other diseases of the respiratory system	0.041	0.075	0.023	0.014
97a	Diseases of the nasal fossae	0.031	0.056	0.051	0.095
106	Pulmonary emphysema	0.031	0	0.021	0
98	Diseases of the larynx	0.017	0	0.014	0.014
97b	Other diseases of the nasal fossae	0.014	0	0.007	0.109
9	Whooping cough	0.003	0	0	0
37a	Acute disseminated tuberculosis	0.003	0	0	0.014
104	Gangrene of the lung	0	0	0.005	0
10	Diphtheria	0	0	0	0.014
	Totals	11.996	7.017	13.074	7.554

TABLE 4

Percentage of Deaths at Ages 90 Years and Over Assignable to the Primary and Secondary Sex Organs. Class III

INTERNAT. LIST NO.	CAUSE OF DEATH	PER CENT OF TOTAL DEATHS			
		Males		Females	
		White	Colored	White	Colored
135	Diseases of the prostate	1.838	1.400	0	0
38	Syphilis	0.014	0.149	0.005	0.082
47	Cancer and other malignant tumors of the breast	0.007	0	0.730	0.435
134	Diseases of the urethra, etc.	0.007	0.056	0.002	0.014
136	Nonvenereal diseases of the male genital organs	0.007	0	0	0
40	Gonococcus infection	0.003	0	0	0
46	Cancer and other malignant tumors of female genital organs	0	0	0.307	0.448
137	Cysts, other benign tumors of ovary	0	0	0.028	0
139	Benign tumors of the uterus	0	0	0.028	0.014
141	Other diseases of the female genital organs	0	0	0.014	0.014
142	Nonpuerperal diseases of the breast	0	0	0.005	0.014
138	Salpingitis and pelvic abscess	0	0	0.009	0
	Totals	1.876	1.605	1.128	1.021

TABLE 5

Percentage of Deaths at Ages 90 Years and Over Assignable to the Kidneys and Related Excretory Organs. Class IV

INTERNAT. LIST NO.	CAUSE OF DEATH	PER CENT OF TOTAL DEATHS			
		Males		Females	
		White	Colored	White	Colored
129	Chronic nephritis	10.588	12.167	8.246	8.805
133	Diseases of the bladder	0.594	0.541	0.105	0.095
128	Acute nephritis	0.252	0.653	0.191	0.367
131	Other diseases of kidneys and annexa	0.211	0.299	0.130	0.054
132	Calculi of the urinary passages	0.024	0.037	0.009	0
	Totals	11.669	13.697	8.681	9.321

TABLE 6

Percentage of Deaths at Ages 90 Years and Over Assignable to the Skeletal and Muscular Systems. Class V

INTERNAT. LIST NO.	CAUSE OF DEATH	PER CENT OF TOTAL DEATHS			
		Males		Females	
		White	Colored	White	Colored
52	Chronic rheumatism	0.089	0.131	0.167	0.217
51	Acute rheumatic fever	0.045	0.429	0.070	0.258
155	Diseases of the bones	0.024	0	0.030	0.014
156	Diseases of the joints	0.007	0	0.005	0
34	Tuberculosis of the vertebral column	0.003	0	0.002	0
158	Other diseases of the organs of locomotion	0.003	0	0.007	0.027
35	Tuberculosis of the joints	0	0	0.007	0
	Totals	0.171	0.560	0.288	0.516

TABLE 7

Percentage of Deaths at Ages 90 Years and Over Assignable to the Alimentary Tract and Associated Organs Concerned in Metabolism. Class VI

INTERNAT. LIST NO.	CAUSE OF DEATH	PER CENT OF TOTAL DEATHS			
		Males		Females	
		White	Colored	White	Colored
11b	Influenza (other and unspecified)	2.091	1.846	2.118	2.582
44	Cancer and other malignant tumors of stomach, liver	0.907	0.411	0.972	0.516
114	Diarrhea and enteritis	0.706	0.914	0.860	0.815
112	Other diseases of the stomach	0.549	0.448	0.695	0.666
43	Cancer and other malignant tumors of the buccal cavity	0.402	0.075	0.151	0.027
118a	Hernia	0.396	0.429	0.147	0.054
118b	Intestinal obstruction	0.338	0.112	0.365	0.231
45	Cancer and other malignant tumors of the peritoneum	0.321	0.112	0.498	0.109
16	Dysentery	0.276	0.839	0.242	0.734
122	Cirrhosis of the liver	0.211	0.317	0.193	0.245
119	Other diseases of the intestines	0.164	0.093	0.144	0.109
111a	Ulcer of the stomach	0.097	0.075	0.079	0.082
124	Other diseases of the liver	0.185	0.075	0.228	0.054
109b	Other diseases of the pharynx, tonsils	0.058	0.037	0.047	0.027
123	Biliary calculi	0.058	0.019	0.123	0.014
111b	Ulcer of the duodenum	0.031	0.019	0.005	0
108	Diseases of the mouth and annexa	0.027	0.056	0.030	0.054
117	Appendicitis and typhlitis	0.037	0.093	0.021	0.041
126	Peritonitis without specified cause	0.027	0.093	0.026	0.014
15	Cholera nostras	0.017	0.019	0.021	0.014
54	Pellagra	0.014	0.299	0.007	0.431
33	Tuberculosis of the intestines and peritoneum	0.010	0.037	0.012	0.054
110	Diseases of the esophagus	0.010	0	0.005	0
1	Typhoid and paratyphoid fever	0.007	0.093	0.002	0.041
125	Diseases of the pancreas	0.003	0	0.009	0.014
68	Chronic poisoning by organic substances	0.003	0.019	0.002	0
121	Hydatid tumor of the liver	0	0	0.002	0
37	Disseminated tuberculosis	0	0	0.002	0
116	Diseases due to other internal parasites	0	0	0	0.014
120	Acute yellow atrophy of the liver	0	0	0.007	0
	Totals	6.945	6.530	7.013	6.932

TABLE 8

Percentage of Deaths at Ages 90 Years and Over Assignable to the Nervous System and Sense Organs. Class VII

INTERNAT. LIST NO.	CAUSE OF DEATH	PER CENT OF TOTAL DEATHS			
		Males		Females	
		White	Colored	White	Colored
75	Paralysis without specified cause	0.651	2.220	0.795	2.160
	Suicide	0.175	0	0.026	0.027
73	Other diseases of the spinal cord	0.102	0.075	0.172	0.177
76	General paralysis of the insane	0.078	0.093	0.040	0.068
77	Other forms of mental alienation	0.048	0.019	0.047	0.041
84	Other diseases of the nervous system	0.182	0.093	0.123	0.122
78	Epilepsy	0.034	0.075	0.028	0
72	Tabes dorsalis	0.020	0.019	0.005	0.014
86a	Diseases of the ear	0.020	0.037	0.012	0
82	Neuralgia	0.024	0.019	0.063	0.014
71a	Simple meningitis	0.014	0	0.014	0
86b	Diseases of the mastoid process	0.014	0.019	0.002	0
70	Encephalitis	0.003	0.019	0.019	0
79	Convulsions	0.003	0.019	0.005	0
85	Diseases of the eye and annexa	0.003	0	0.012	0
32	Tuberculosis of the meninges and central nervous system	0.003	0	0	0
66	Alcoholism	0.003	0.019	0	0
71b	Nonepidemic cerebrospinal meningitis	0.003	0	0	0
20	Leprosy	0	0	0.002	0
81	Chorea	0	0	0.005	0
	Totals	1.380	2.726	1.370	2.623

TABLE 9

Percentage of Deaths at Ages 90 Years and Over Assignable to the Skin. Class VIII

INTERNAT. LIST NO.	CAUSE OF DEATH	PER CENT OF TOTAL DEATHS			
		Males		Females	
		White	Colored	White	Colored
48	Cancer and other malignant tumors of the skin	0.894	0.037	0.758	0.082
21	Erysipelas	0.171	0.037	0.146	0.027
152	Furuncle	0.027	0	0.009	0
153	Acute abscess	0.027	0	0.021	0.014
154	Other diseases of the skin and annexa	0.051	0.093	0.112	0.095
6	Smallpox	0.003	0.075	0.005	0
	Totals	1.173	0.242	1.051	0.218

TABLE 10

Percentage of Deaths at Ages 90 Years and Over Assignable to the Endocrinal System. Class IX

INTERNAT. LIST NO.	CAUSE OF DEATH	PER CENT OF TOTAL DEATHS			
		Males		Females	
		White	Colored	White	Colored
57	Diabetes mellitus	0.293	0.336	0.312	0.244
63	Diseases of the adrenals	0.014	0	0	0
60b	Other diseases of the thyroid gland	0.003	0	0.007	0.014
60a	Exophthalmic goiter	0	0	0.014	0
	Totals	0.310	0.336	0.333	0.258

TABLE 11

Percentage of Deaths at Ages 90 Years and Over Not Assignable to Any Particular Organ System. Class X

INTERNAT. LIST NO.	CAUSE OF DEATH	PER CENT OF TOTAL DEATHS			
		Males		Females	
		White	Colored	White	Colored
164	Senility	12.758	29.763	14.007	32.160
	All external causes (except suicide)	4.294	2.370	6.546	2.607
49	Cancer and other malignant tumors of other or unspecified organs	0.928	0.522	0.477	0.231
204 and 205	Ill-defined diseases	0.665	8.397	0.746	8.533
69	Other general diseases	0.075	0.075	0.053	0.054
36	Tuberculosis of other organs	0.014	0.019	0.005	0.014
50	Tumors, benign and unqualified	0.007	0	0.044	0.027
25	Other epidemic and endemic diseases	0.006	0.019	0.002	0.041
	Totals	18.747	41.165	21.880	43.667

The totals of Tables 2-11 inclusive sum up to 100 per cent, in each of the race-sex columns. In other words, all of the deaths of persons aged 90 years or over at the time of their demise, during the years 1923-1927 inclusive, are accounted for in these tables. The totals are shown graphically in Fig. 1.

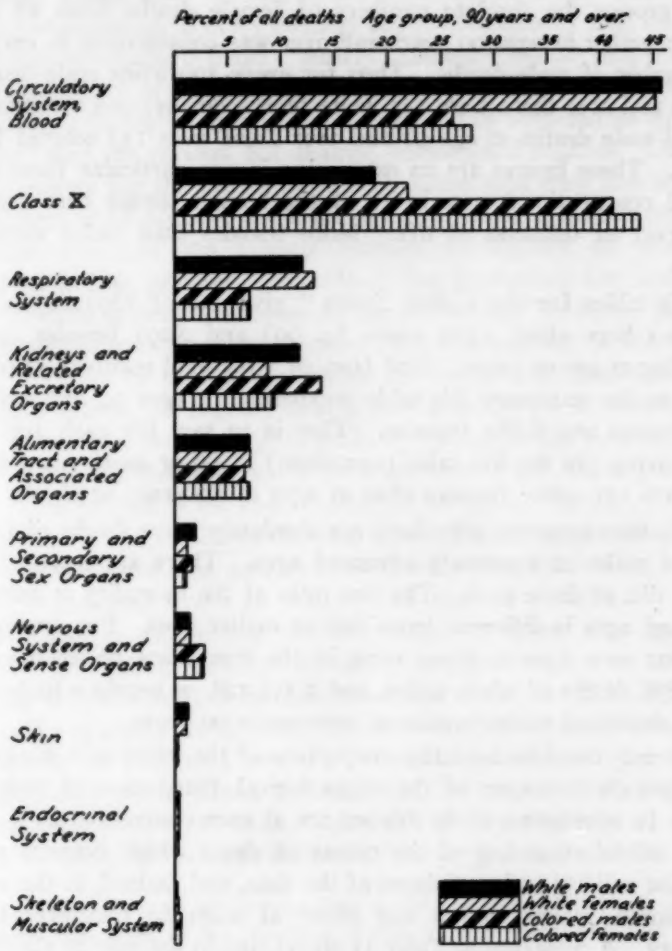

FIG. 1. DIAGRAM SHOWING THE RELATIVE IMPORTANCE OF THE DIFFERENT ORGAN SYSTEMS IN THE MORTALITY AT AGES 90 YEARS AND ABOVE, IN THE U. S. REGISTRATION AREA, 1923-1927. DATA FROM TABLES 2-11 INCLUSIVE

IV

We may turn now to the discussion of the observations which have been set forth above.

The first point of constitutional interest to be noted is brought out by the figures of Table 1. It is seen that for both white and colored racial groups the absolute numbers of female deaths from all causes taken together at ages 90 years and over was considerably in excess of the number of male deaths. Thus for every 100 white male deaths at age 90 and over there were 147 white female deaths; and for every 100 colored male deaths at age 90 and over there were 137 colored female deaths. These figures are an expression in one particular form of the normal constitutional superiority of the human female over the male in respect of duration of life. More females than males survive to age 90.

Life tables for the United States [26] give, out of 100,000 whites of each sex born alive, 1,523 males (p. 69) and 2,291 females (p. 75) surviving at age 90 years. And (*loc. cit.*) the total numbers of persons living in the stationary life table population, at ages 90 and over, are 4,553 males and 6,881 females. That is to say, for each 100 white males living (in the life table population) at 90 or more years of age, there are 151 white females alive at ages of 90 years or above.

It is thus apparent why there are absolutely more deaths of females than of males at extremely advanced ages. There are more of them left to die, at those ages. The sex ratio of the mortality at extremely advanced ages is different from that at earlier ages. For example, to take our own figures, there were in the five years under discussion 2,781,878 deaths of white males, and 2,351,228, or nearly a half-million *fewer*, deaths of white females at ages *under* 90 years.

We may consider next the *comparison of the white with the colored racial groups* in respect of the organological distribution of their mortality. In attempting to do this we are at once confronted by a defect in the official recording of the causes of death which detracts greatly from the validity and usefulness of the data, and, indeed, in the case of the colored groups, makes any effort at scientific treatment largely nugatory. A reference to Table 11 shows that in the case of the colored males approximately 30 per cent, and in the case of the colored females

[26] Glover, J. W. United States Life Tables, 1890, 1901, 1910, and 1901-1910. *Bureau of the Census, Washington* (Gov't Printing Office), 1921.

approximately 32 per cent *of all deaths* at ages of 90 and over were recorded as due to *senility*. But in spite of the fact that Terence (*Phormio, Act IV., Sc. I., 9*) makes Chremes say "Senectus ipsa est morbus" "senility" is, of course, neither a disease nor a "cause of death" in any proper pathological sense. It is merely a piece of medievalism preserved, decade after decade, in the International List. The history of death registration in every country shows conclusively that the number of deaths classified under "senility" declines just in proportion as the whole system of registration is vigorously and intelligently administered. The biological fact is that these persons recorded as dying of "senility" died for various, but definite, pathological reasons. They did not die merely for the purely numerical reason that they had accumulated a store of 90 or more years apiece. Furthermore, it is an observed fact that wherever the general average intelligence of practicing physicians is low, and the standards of the profession are low, there the proportion of deaths recorded as due to "senility" is high. Just this state of affairs prevails relative to the colored population of the United States. Except in the large cities of the north, and even there to a deplorable degree, the negroes usually and normally get a relatively low grade of medical service. This probably accounts for practically the whole of the difference between the colored and the white groups in Table 11, in respect of the deaths recorded as due to "senility."

But from the viewpoint of the vital statistician the trouble lies further back than this. As long as they are officially permitted to do so physicians will record deaths as due to "old age" or "senility." In the United States at the present time they begin doing so after age 60 is reached. The rubric "senility" should be dropped, at once and forever, from the official International List of causes of death. Scientifically it never had any status and belongs with those ancient women, who, in the days of the early bills of mortality, acted as "viewers of the dead."[27] Philosophically it has much the same kind of content and degree of exactness that such a rubric as "Failure to live" would have as a cause of death. Actually the International List *has* just such a rubric as the

[27] Some fourteen years ago a committee of distinguished medical men and vital statisticians, appointed by the American Public Health Association and working under the chairmanship of Dr. Haven Emerson had the following to say about "senility" as a cause of death (*Public Health Repts.*, Vol. 32, p. 1620. Sept. 28, 1917): "Senility. 1. The committee considers this a very unsatisfactory title and not reliable as a cause of death, without autopsy. It should always be queried for the *disease causing death*, as it is too often used on death certificates of elderly per-

latter in its "Ill-defined diseases." "Ill-defined diseases" tells only that the maker of the death certificate did not know what the unfortunate victim died of, and presumably was not inclined to take any trouble to find out. It will be noted again from Table 11 that "Ill-defined diseases" are blamed for a *ten times* higher percentage of nonagenarian and centenarian deaths among the colored than among the whites. The same result appears if we examine death rates instead of death ratios. Official figures for the Registration States in 1927[28] give for "Ill-defined diseases" a death rate, per 100,000 estimated population, of 8.2 in the case of whites and 91.7 in the case of colored. This is another indication of the poorer average quality of medical service among the colored than among the whites.

It would seem desirable that at the next revision of the International List some consideration should be given to the elimination of these two rubrics, "Senility" and "Ill-defined diseases." If nothing better can be done it would at least be more honest and decent intellectually to dump these deaths together into a category labeled "Cause of death unknown," and then bend every effort to reducing the number of entries under such a rubric.

With the above prefatory remarks in mind we may turn to the detailed examination of the summarized data presented in Table 12.

The organ systems are arranged in Table 12 in descending order of the percentages for white males. The white female column preserves the same order except that Classes III and VII are interchanged. But the order of the classes is considerably changed in the colored groups. This arises in large part presumably because of relatively huge percentages in Class X for the colored, the reasons for which have already been discussed.

An examination of Table 12 suggests that the greater portion of the colored deaths recorded in Table 11 as due to "Senility" and "Ill-defined diseases" probably really belonged to Classes I (Circulatory system) and II (Respiratory system). Fig. 1 indicates this point

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sons whose deaths should have been reported as due to diseases of various organs." The words "without autopsy" in the statement quoted must surely have been inadvertent, because "senility" is in no better case as a "cause" title *with* an autopsy than it is *without* one. One would like enormously to see autopsy findings which would justify it as a cause of death!

[28] Mortality Statistics: 1927. Part II. Text and Text Tables. *Bureau of the Census, Washington, D. C.*, 1930. (*Cf.*, Table I, p. 22.)

TABLE 12

Showing the Relative Importance of Different Organ Systems in the Mortality at Ages 90 Years and Over in the U. S. Registration States, 1923-1927

CLASS NO.	ORGAN SYSTEM	PER CENT OF TOTAL DEATHS			
		Males		Females	
		White	Colored	White	Colored
I.	Circulatory system, blood	45.732	26.124	45.182	27.893
II.	Respiratory system	11.996	7.017	13.074	7.554
IV.	Kidneys and related excretory organs	11.669	13.697	8.681	9.321
VI.	Alimentary tract and associated organs	6.945	6.530	7.013	6.932
III.	Primary and secondary sex organs	1.876	1.605	1.128	1.021
VII.	Nervous system and sense organs	1.380	2.726	1.370	2.623
VIII.	Skin	1.173	0.242	1.051	0.218
IX.	Endocrinal system	0.310	0.336	0.333	0.258
V.	Skeletal and muscular system	0.171	0.560	0.288	0.516
	Total deaths classifiable by organ systems	81.252	58.837	78.120	56.336
X.	Not capable of organological classification	18.747	41.165	21.880	43.667

clearly. The only other important organological class (among nonagenarians), namely IV (Kidneys, etc.) shows a *higher* percentage of total mortality in the colored than in the white.

Purely as a tentative procedure it may be assumed that the colored excess over the whites in Class X may be prorated to Classes I and II in the same proportions as the deaths actually assigned to those two classes among the colored. If this is done we get 43.754 per cent for Class I, colored males, and 45.105 per cent for Class I, colored females. These figures are nearly the same as the corresponding figures for whites. In the same way, on the assumption stated, we get 11.704 for the colored male percentage in Class II, and 12.129 for the corresponding percentage for the colored females. These again are nearly the same as the observed white percentages, but slightly lower. These "corrections," however, can only be regarded as purely speculative, and warrant no conclusions. We know of no data available at present from which an unequivocal judgment may be reached as to the true comparative mortality by organ systems of the colored and white at ages of 90 years and over.

It is an interesting observation, in connection with these tentative "corrections" that the proportionate relations between the recorded percentage mortalities in Class I and Class II in Table 12 are closely simi-

lar for colored and white groups. Thus the percentage of all deaths assigned to class I (Circulatory system) is 3.8 times as great as the percentage of all deaths assigned to Class II (Respiratory system) in the case of white males. In the case of colored males it is 3.7 times. Turning to the females a similar result appears. The recorded percentage of all deaths in Class I is 3.5 times the recorded percentage in Class II in the case of white females, and 3.7 times in the case of colored females.

While this is suggestive it is not probative. Still it may possibly be the fact that the great apparent biological inferiority of the circulatory and respiratory systems of the negro as compared with the white, when all ages are considered together,[29] does not hold after age 90. Instead it may be that the colored who survive to 90 have these organ systems about as sound as do whites who survive to the same age.

As has already been suggested above, it may probably be safely inferred that, at these extremely advanced ages, death associated with breakdown of the kidneys and related excretory organs occurs with really greater proportionate frequency among the colored than among the whites. The same thing is probably true of the mortality associated with the skeletal and muscular systems.

On the face of the figures it would appear that the superiority of the colored skin to the white in resistance to fatal diseases, which has been discussed by Holmes [30] extends over into these advanced ages. But for the reasons set forth above the figures cannot be taken at their face value.

Confining attention, from this point on, to the whites it appears that the two *sexes* are not significantly different, at these advanced ages of 90 years and above, in respect of the percentage of deaths assignable respectively to the circulatory system, the alimentary tract, the nervous system, the skin, and the endocrine system. But the females have a distinctly smaller percentage of all deaths than do the males assigned to the respiratory system, to the kidneys, and to the primary and sec-

[29] *Cf.* Pearl's study of autopsy and other data (HUMAN BIOLOGY, Vol. I, pp. 229-249, 1929) and official death rates, which, for example, give (*loc. cit. supra*) in 1927 a respiratory system (as defined in the International List) rate per 100,000 of 89.3 in whites and 175.2 in the colored, and a circulatory system (as defined in the International List) rate of 230.7 in the whites as against 250.2 in the colored.

[30] Holmes, S. J. The resistant ectoderm of the negro. *Amer. Jour. Phys. Anthrop.*, Vol. 12, pp. 139-153, 1928; Differential mortality in the American negro. HUMAN BIOLOGY, Vol. III, pp. 71-106; 203-244, 1931.

ondary sex organs. The strains of child bearing upon the reproductive organs and the kidneys are long past in the woman of 90; the man of that age is still liable to prostatic difficulties.

So far attention has been directed exclusively to the nonagenarian (plus alleged centenarians) group. It is now desirable to compare the organological distribution of mortality in this group with that of adults in other narrowly delimited age groups. To this end Table 13 has been prepared. The data were extracted from the same sources, and for the same years, as Tables 1-12 inclusive. Three age groups were chosen for comparison. These were 20-24 years, 40-44 years, and 60-64 years. Thus we have the young adults just at the end of the growth period of life; the middle-aged persons at the so-called "prime of life"; and finally the persons past middle age but not yet quite in the "old age" category. Because of the difficulty noted in the preceding section only deaths of whites were extracted, no further attention being paid to the colored.

Considering first the mortality assignable to Class I (Circulatory system, blood, etc.) it is apparent that it includes a steadily increasing proportion of all deaths as age advances, until in those dying at ages of 90 years and over it is responsible for nearly one-half the total mortality in both sexes, a much larger proportion of the total mortality at those ages than can be assigned to any other single organ system. The trend is shown graphically in Fig. 2.

Pearl [31] has shown that, from age 60 on to the end of the life span, the death *rate* assignable to breakdown of the circulatory system is higher than that associated with any other organ system. It is now seen that the *proportion* (death ratio) of deaths associated with the circulatory system is also higher than that associated with any other organ system at the extremely advanced ages under discussion. It appears that the circulatory system, more often than any other part of the body, by its eventual failure to meet the biological demands made upon it sets the upper limit of the human life span.

Both the specific death rates and the death ratios for Class I are closely similar in their values and trends in males and females.

The mortality associated with the respiratory system (Class II) presents a somewhat different picture. The percentage of the total deaths assignable here is not essentially different at ages of 90 years

[31] Pearl, R. The Biology of Death. *Philadelphia* (J. B. Lippincott Co.), 1922. Chap. IV, p. 115.

and over, from what it is in the 60-64 year group, as is shown in Fig. 3 and Table 13, and there is no marked difference between the sexes in this regard. Roughly somewhere from 10 to 13 per cent of all the deaths at these ages from 60 on to the end of life are attributable to causes associated with the respiratory organs. At the beginning of adult life the respiratory system stands at the top of the list (except for Class X to be discussed later) with 30 to 40 per cent of all deaths assignable to it. The 40-44 year group shows smaller percentages and a marked alteration in the position of the two sexes, the female per-

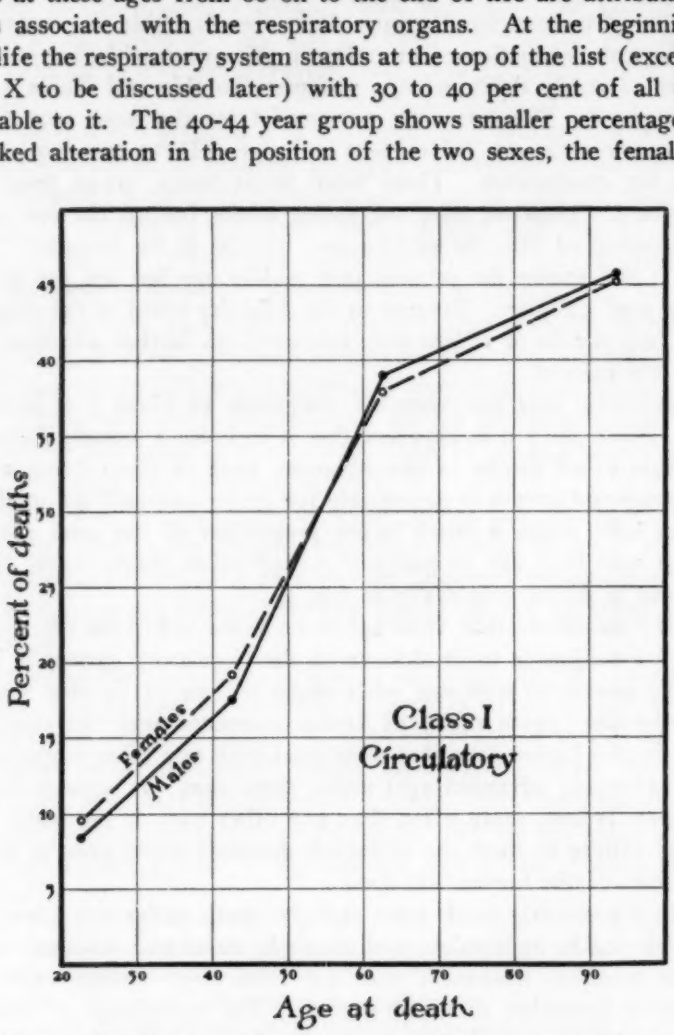

FIG. 2. SHOWING THE CHANGES IN THE PERCENTAGE OF TOTAL DEATHS ASSIGNABLE TO CLASS I (CIRCULATORY SYSTEM, BLOOD, ETC.) WITH ADVANCING AGE, IN ADULT WHITE MALES AND FEMALES. DATA FROM TABLE 13

centage now being lower than the male, just as it was markedly higher than the male percentage in the 20-24 group. A similar relation is shown by the age specific death rates for Class II mortality (*cf.*, Pearl, *loc. cit.*, pp. 115 and 120). From ages 10 to 20 years the female death rate curve lies above the male curve, while from roughly 30 to 60 years the female respiratory mortality is markedly lower than the male rate. Why the female should be freer than the male from fatal pulmonary breakdown in early middle life is not clear. Possibly it is a reflection of occupational differences, as was suggested in the earlier discussion referred to, but the case is not clear.

The changes with age in the proportionate mortality attributable to the sex organs are shown in Fig. 4.

Among the males only a relatively small percentage mortality is

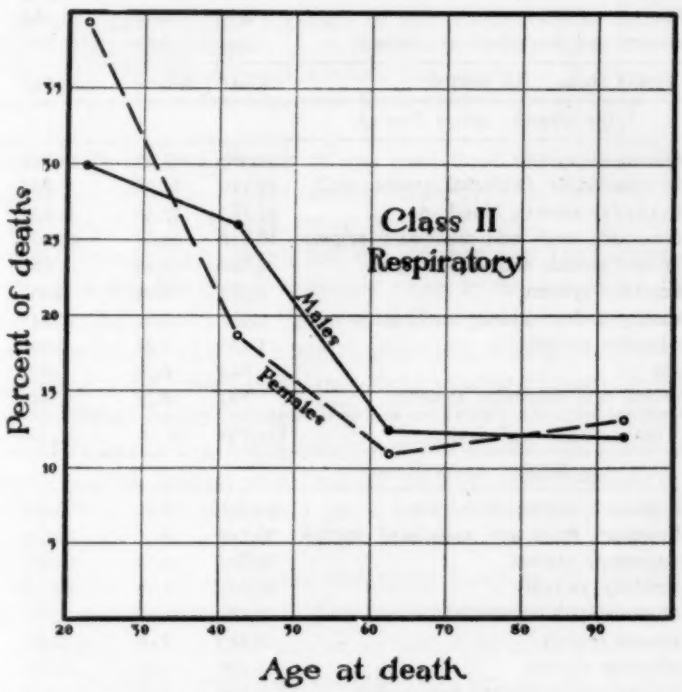

FIG. 3. SHOWING THE CHANGES IN THE PERCENTAGE OF TOTAL DEATHS ASSIGNABLE TO CLASS II (RESPIRATORY SYSTEM) WITH ADVANCING AGE, IN ADULT WHITE MALES AND FEMALES. DATA FROM TABLE 13

TABLE 13

Absolute and Percentage Frequency of Mortality Assignable to Different Organ Systems in Three Adult Age Groups Among White Persons in the U. S. Registration States, 1921-1927 Inclusive

	ORGAN SYSTEM	MALES		FEMALES	
	(Age Group. 20-24 Years)	Absolute	Percentage	Absolute	Percentage
X.	Not classifiable (external causes, etc.)	23,285	33.34	5,213	7.44
II.	Respiratory system	20,871	29.88	26,830	38.30
VI.	Alimentary tract and associated organs	9,578	13.71	11,682	16.67
VII.	Nervous system and sense organs	5,978	8.56	4,141	5.91
I.	Circulatory system, blood, etc.	5,865	8.40	6,757	9.65
IV.	Excretory system	1,895	2.71	2,304	3.29
V.	Skeletal and muscular system	774	1.11	576	0.82
IX.	Endocrine system	772	1.11	1,319	1.88
VIII.	Skin	435	0.62	360	0.52
III.	Primary and secondary sex organs	394	0.56	10,875	15.52
	Totals 20-24. All deaths	69,847	100.00	70,057	100.00
	(Age Group. 40-44 Years)				
II.	Respiratory system	31,489	25.99	17,127	18.73
X.	Not classifiable (external causes, etc.)	23,120	19.08	5,855	6.41
I.	Circulatory system, blood, etc.	21,183	17.48	17,534	19.18
VI.	Alimentary tract and associated organs	18,017	14.87	16,025	17.53
VII.	Nervous system and sense organs	14,822	12.24	5,681	6.21
IV.	Excretory system	7,563	6.24	8,008	8.76
III.	Primary and secondary sex organs	1,937	1.60	17,147	18.76
IX.	Endocrine system	1,481	1.22	3,001	3.28
VIII.	Skin	844	0.70	505	0.55
V.	Skeletal and muscular system	704	0.58	543	0.59
	Totals 40-44. All deaths	121,160	100.00	91,426	100.00
	(Age Group. 60-64 Years)				
I.	Circulatory system, blood, etc.	87,101	38.99	66,428	37.88
VI.	Alimentary tract and associated organs	36,145	16.18	28,855	16.46
II.	Respiratory system	27,809	12.45	19,083	10.88
IV.	Excretory system	25,029	11.20	20,671	11.79
X.	Not classifiable (external causes, etc.)	23,189	10.38	9,886	5.61
VII.	Nervous system	12,249	5.48	5,464	3.12
IX.	Endocrine system	5,506	2.47	9,870	5.63
III.	Primary and secondary sex organs	3,800	1.70	13,295	7.59
VIII.	Skin	1,654	0.74	925	0.53
V.	Skeletal and muscular system	907	0.41	888	0.51
	Totals 60-64. All deaths	223,389	100.00	175,365	100.00

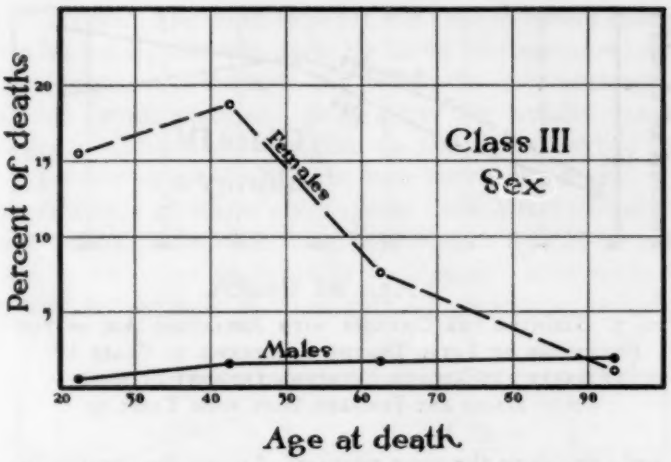

FIG. 4. SHOWING THE CHANGES IN THE PERCENTAGE OF TOTAL DEATHS ASSIGNABLE TO CLASS III (PRIMARY AND SECONDARY SEX ORGANS) WITH ADVANCING AGE, IN ADULT WHITE MALES AND FEMALES. DATA FROM TABLE 13

associated with the sex organs at any period of adult life. The percentage is only slightly higher among those dying at ages of 90 years and over than it is in the age group 60-64, or 40-44. The death rates for Class III males (*cf.*, Pearl, *loc. cit.*, pp. 115 and 125) make a more marked and a steadier ascent from age 20 on to the end of the life span than do the death ratios. In the case of the females the percentages of deaths assignable to Class III are considerable during the reproductive period, rising from about 15 in the 20-24 year age group to nearly 19 per cent in the 40-44 age group. With the passing of the menopause the percentage falls rapidly, being 7.6 in the 60-64 year age group (where cancer of uterus, breast, and ovaries are the principal causes), and reaches the low level of about 1 per cent in those dying at ages of 90 years and over.

The percentage of all deaths assignable to Class IV (Kidneys and related excretory organs) is higher among those dying at ages of 90 years and above than it is at any other period of adult life, as is shown in Fig. 5. The male curve rises steadily from age 20 on. This parallelism to what is seen in Fig. 2 for the circulatory system is perhaps what might be expected in view of the close connections which exist between renal and vascular pathology. The death rate curves (*cf.*, Pearl, *loc. cit.*,

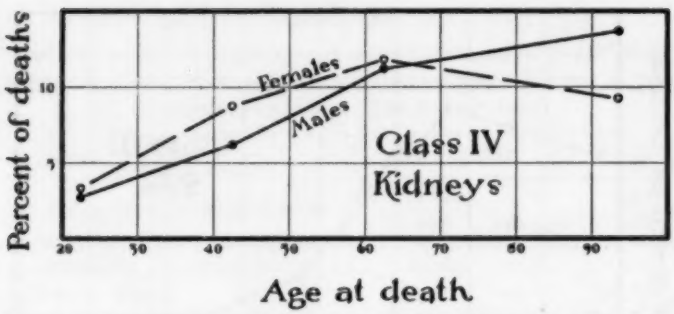

FIG. 5. SHOWING THE CHANGES WITH ADVANCING AGE IN THE PERCENTAGE OF TOTAL DEATHS ASSIGNABLE TO CLASS IV (KIDNEYS AND RELATED EXCRETORY ORGANS) IN ADULT WHITE MALES AND FEMALES. DATA FROM TABLE 13

pp. 115 and 127) show the same relation. Among the females the death ratio curve for Class IV roughly parallels the male curve, and lies above it up to the age group 60-64. The very old females (dying at 90 years and over) exhibit a lower percentage of deaths attributable to Class IV than do the males in the same age group, and lower than the females of the 60-64 year age group.

The very old group dying at ages of 90 years and over exhibits only a small percentage of deaths attributable to the alimentary tract and its associated organs (Class VI), less than a third of the percentage at

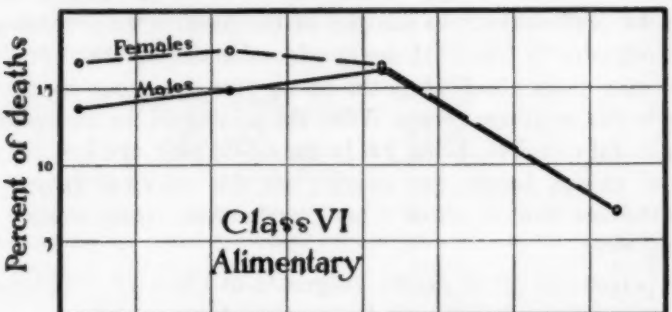

FIG. 6. SHOWING THE CHANGES WITH ADVANCING AGE IN THE PERCENTAGE OF TOTAL DEATHS ASSIGNABLE TO CLASS VI (ALIMENTARY TRACT AND ASSOCIATED ORGANS) IN ADULT WHITE MALES AND FEMALES. DATA FROM TABLE 13

earlier adult ages. The death ratio for this class of causes rises a little, but probably not significantly, from the 20-24 year age group up to the 60-64 year group, as is shown in Fig. 6. In the early adult age groups here studied (20-24 years and 40-44 years) the females show higher percentages of alimentary deaths than the males. At the two later age groups the percentages are nearly the same for the two sexes.

The percentage of deaths assignable to Class VII (Nervous system and sense organs) is lower in the group dying at ages of 90 years and over than in any other adult age group studied. It is lower for the females than for the males in all of the four age groups, as is shown in Fig. 7.

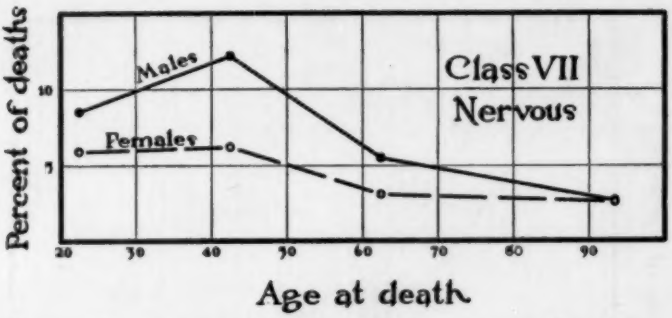

FIG. 7. SHOWING THE CHANGES WITH ADVANCING AGE IN THE PERCENTAGE OF TOTAL DEATHS ASSIGNABLE TO CLASS VII (NERVOUS SYSTEM AND SENSE ORGANS) IN ADULT WHITE MALES AND FEMALES. DATA FROM TABLE 13

The mortality percentage assignable to the nervous system is higher in the 40-44 year age group than in the 20-24 age group, considerably so for the males and slightly so for the females. At age 60-64 years the percentage has dropped to a point below that for the 20-24 age group, and the decline continues to the nonagenarian group as already noted.

Classes V (Skeletal and muscular system) and VIII (Skin), play such a small rôle numerically in the total mortality at any age that detailed discussion of them here is not necessary. Class IX (Endocrine system) shows a steadily rising percentage of deaths up to the age group 60-64 years, reaching something over 5 per cent of all deaths in the females of that age group, but then drops off to an insignificant figure in both sexes of the 90 years and over group.

We come now to the consideration of Class X, the mortality not

classifiable on an organological basis. As shown in Fig. 8, the percentage mortality in this class declines from age 20-24 to age 60-64, and then rises to relatively high values in the 90 and over age group. The male curve starts at a very high value in the 20-24 year group, where a third of all the deaths within the age group fall in this class. The female curve starts with the low value of 7.4 per cent in the 20-24 year group and declines only slightly to a value of 5.6 per cent in the 60-64 year group, but rises sharply to a value nearly four times as great in the 90 years and over group. The rise of the male curve after the age group 60-64 is not so marked, the final value being about the same as that for the 40-44 year group.

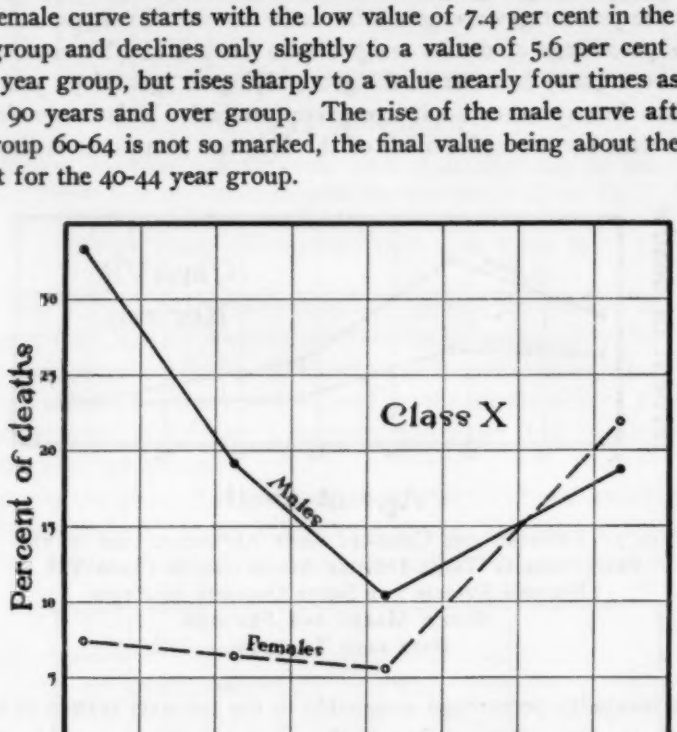

FIG. 8. SHOWING THE CHANGES WITH ADVANCING AGE IN THE PERCENTAGE OF TOTAL DEATHS ASSIGNABLE TO CLASS X (MORTALITY NOT ORGANOLOGICALLY CLASSIFIABLE) IN ADULT WHITE MALES AND FEMALES. DATA FROM TABLE 13

Since this Class X includes such a large proportion of the total mortality in all the age groups studied among males, and in the final one among females, it is desirable to examine more carefully into its composition. As the raw data are furnished by official tabulations classi-

fied according to the International List there are two noteworthy "causes" which fall into Class X because of their insufficient subdivision. These are "36. Tuberculosis of other organs"; and "49. Cancer of other and unspecified organs."

Two other "cause" groups in the International List make considerable contributions to our Class X because they are badly defined catch-alls with dubious or no biological significance. These are "XIII. Old age (senility)"; and "XV. Ill-defined diseases." These have already been discussed above.

Finally a very large contribution to our Class X is made by Class XIV of the International List, External causes (here taken *less* suicide). This is a perfectly logical class, and is unclassifiable organologically solely because of the nature of the case, and not because of any defect of the International List.

The magnitude of the contribution of each of these six omnibus "causes" to the total mortality in our Class X is shown in the figures assembled in Table 14. The other "causes" which are included in our Class X besides the five mentioned make such insignificant contributions to the total in the class that they may be neglected in this discussion.

From Table 14 it appears that:

1. "Tuberculosis of other organs" makes so small a part of the total of Class X in any of the four age groups that it cannot sensibly affect any results or conclusions.

2. While making a somewhat larger contribution "Ill-defined diseases," in the white group here dealt with, again is not numerically a very important rubric.

3. In the 20-24 year, and the 40-44 year groups, among the males, the "External causes *less* suicide" item is overwhelmingly preponderant numerically in making up the totals of our Class X. The same is true of the females in the age group 20-24. Thus it appears (referring back to Table 13) that in the age group 20-24 "External causes *less* suicide," that is to say *traumatic accidents,* constitute the most important cause category of Class X of deaths for males.

4. In the age group 60-64 in the males, and the two age groups 40-44 and 60-64 in the females "Cancer of other and unspecified organs" comes forward as a numerically important element in the Class X totals.

5. Finally in the age group 90 years and over, deaths recorded as due to "senility" account for more than 60 per cent of the totals in

TABLE 14

Showing the Contribution to Our Class X of Certain "Causes" of the International List, in Four Age Groups. Whites Only. Registration States, 1923-1927

AGE AND SEX GROUPS	TUBERCULOSIS OF OTHER ORGANS		CANCER OF OTHER ORGANS		OLD AGE		ILL-DEFINED DISEASES		EXTERNAL CAUSES *less* SUICIDE	
	Abs. No. of Deaths	Per cent of Total in Class X	Abs. No. of Deaths	Per cent of Total in Class X	Abs. No. of Deaths	Per cent of Total in Class X	Abs. No. of Deaths	Per cent of Total in Class X	Abs. No. of Deaths	Per cent of Total in Class X
Males										
Age group 20-24 years	191	0.82	603	2.59	...	...	238	1.02	22,130	95.04
Age group 40-44 years	264	1.14	2229	9.64	...	...	599	2.59	19,859	85.90
Age group 60-64 years	145	0.63	7904	34.09	189	0.82	1482	6.39	13,205	56.95
Age group 90 and over years	4	0.07	272	4.95	3740	68.05	195	3.55	1,259	22.91
Females										
Age group 20-24 years	181	3.47	342	6.56	...	...	272	5.22	4,288	82.26
Age group 40-44 years	181	3.09	1522	25.99	...	...	379	6.47	3,594	61.38
Age group 60-64 years	116	1.17	3923	39.68	240	2.43	751	7.60	4,567	46.20
Age group 90 and over years	2	0.02	205	2.18	6023	64.01	321	3.41	2,815	29.92

Class X, nearly all the rest being made up by "External causes *less* suicide."

If registration authorities would, in the first place, abandon "senility" entirely as a recognized cause of death, and refuse to accept death certificates bearing it as a primary cause; and then refuse to accept any death certificate for cancer at any age which failed to state the organ affected, a really significant improvement in vital statistics would have been made.

VI

The structural and functional organization of the vertebrate body is such that certain organ systems come normally and regularly into direct and immediate contact with the external environment, while other organ systems do not, but are, on the contrary, protected from such contact. On this basis the organ systems may be classified as follows:

A.	B.
Organ systems coming into direct contact with the external environment	*Organ systems not coming into direct contact with the external environment*
Respiratory system	Circulatory system
Sex organs	Skeletal and muscular system
Kidneys and associated excretory organs	Nervous system
Alimentary tract	Endocrine system
Skin	

Little comment on this classification is necessary. The respiratory system and the alimentary tract are obviously in direct contact with environmental objects and material all the time. The primary and secondary sex organs are either directly exposed to environmental influences, or are accessible with greater or less ease to infectious agents through ducts or tubes. The breasts are particularly exposed to external traumatic injury with no other protection than the skin, and are subject to infection from external sources through the ducts of the nipples.

On the other hand the organ systems in the B column present a different picture. The circulatory system is a wholly internal closed system which nowhere comes *directly* into contact with the external world under normal circumstances. The same is true of the skeleton and muscles. Certain of the latter, to be sure, have only the skin as

protection against external traumatic injury, but still it remains true that everywhere there is something (skin or mucous membrane) between muscles and the external world. These tissues when in their normal unbroken state are a highly effective protective mechanism against infection from external sources. The nervous system is obviously well protected against direct contact with the external environment. The thyroid, pituitary, and adrenal glands, and the islands of Langerhans are normally never in contact with, nor accessible to direct contact with the external environment.

It will now be of interest to see how the mortality of whites is distributed between these A and B groups, at the four different adult age periods here under discussion. This will be considered from the standpoint of death ratios in Table 15. To this table there is added, as a final column, the percentages in Class X (Deaths not organologically classifiable) in order that it may be available for reference.

TABLE 15

Percentage of Total Mortality at Four Age Periods Assignable to (A) Organs in Direct Contact With the External Environment, to (B) Organs Not in Direct Contact with the External Environment, and to (C) Class X. Whites Only. Registration States, 1923-1927

SEX AND AGE GROUPS	A DIRECT CONTACT	B NO DIRECT CONTACT	C CLASS X
Males	*Per cent*	*Per cent*	*Per cent*
Age group 20-24 years	47.48	19.18	33.34
Age group 40-44 years	49.40	31.52	19.08
Age group 60-64 years	42.27	47.35	10.38
Age group 90 and over years	33.67	47.59	18.75
Females			
Age group 20-24 years	74.30	18.26	7.44
Age group 40-44 years	64.33	29.26	6.41
Age group 60-64 years	47.25	47.14	5.61
Age group 90 and over years	30.94	47.17	21.88

Because of the small and fairly steady percentages in Class X in each of the first three age periods for the females in Table 15, the general trend of the results is clearer than for the males. In the case of the females the percentage of deaths attributable to the A group of organs (direct contact with the external environment) is *highest* at ages 20-24 years, and is successively smaller at each older age group studied,

until finally the *Greisen* dying at 90 years and over have only about 31 per cent of their total mortality chargeable against these organs. Correspondingly the percentage of deaths attributable to the B group of organs (no direct contact with the external environment) is *lowest* at ages 20-24, and increases in each later age group. In the final group (90 years and over) the case is complicated by the presence of the relatively large percentage in Class X, which, as we have already seen, is among the females mainly due to deaths recorded under "Senility." It is probable that most of these would really fall, if these were correctly recorded, among the B group of organs, especially the circulatory system. But even without attempting now to make any numerical correction for this evident source of error, a general trend is plainly suggested by the figures for the females in Table 15. *With advancing adult age the proportion of deaths due to causes having their pathological lesions or clinical manifestations associated with the organ systems of the body which are normally and regularly in direct contact with the external environment decreases, while the proportion having their pathological lesions or clinical manifestations associated with the organ systems normally protected from direct contact with the external environment increases.*

The same relationship probably holds for the males but is obscured by the relatively high proportion of deaths in Class X due to traumatic accidents in the three earlier age periods.

Looking at the whole matter biologically it is reasonable to regard deaths due to traumatic accidents ("External causes except suicide" of the International List) as mortality due to fatal impacts of the environment upon the organism as a whole, rather than upon a single organ system, as is commonly the case with those fatal impacts which are due to infection by pathogenic micro-organisms. Purely as a tentative and hypothetical groping after insight into a complex and difficult problem it will be interesting to construct from Table 15 a new theoretical Table 16, in which to Column A as it stands in Table 15 are added the percentages of deaths at each age due to "External causes except suicide"; and to Column B the percentages of deaths at each age recorded as due to "Senility," because there is good reason to believe that the great majority of such deaths, if correctly determined, would be found to be connected with failure of the circulatory system (diseases of the heart or the arteries). The result, as will be seen from the table and from Fig. 9, is strikingly regular.

TABLE 16

Theoretical Table of Percentages of Total Mortality Constructed as Described in Text

SEX AND AGE GROUP	A DIRECT CONTACT PLUS TRAUMATIC IMPACT OF ENVIRONMENT	B NO DIRECT CONTACT PLUS "SENILITY"	RESIDUE LEFT IN CLASS X
	Per cent	*Per cent*	*Per cent*
Males			
Age group 20-24 years	79.16	19.18	1.66
Age group 40-44 years	65.79	31.52	2.69
Age group 60-64 years	48.18	48.20	3.62
Age group 90 and over years	37.96	60.35	1.69
Females			
Age group 20-24 years	80.42	18.26	1.32
Age group 40-44 years	68.26	29.26	2.48
Age group 60-64 years	49.85	47.28	2.86
Age group 90 and over years	37.49	61.18	1.33

If it is remembered that the only thing which is "theoretical" about Table 16 is relative to the *grouping* of actually observed and officially recorded statistics of death, it is clear that there is a quite remarkable regularity exhibited in the results.

In the first place Class X is reduced to practically negligible proportions in all four adult age groups studied.

In the second place the quantitative similarity between male and female proportionate mortality is striking in all four adult age groups studied. Furthermore it is noteworthy that the proportionate mortality associated with the A group of organ systems is *higher* (by small amounts) in the females than in the males at each of the first three adult age periods studied; while in the case of the B group of organ systems the female percentages are *lower* (by small amounts) than the male percentages at each of the first three age periods studied. In the final age period these relations are reversed but the differences between the sexes in this group are so small as to be presumably wholly insignificant.

In the third place the rule of the *decline* with advancing adult age of the percentage of mortality associated with organ systems normally

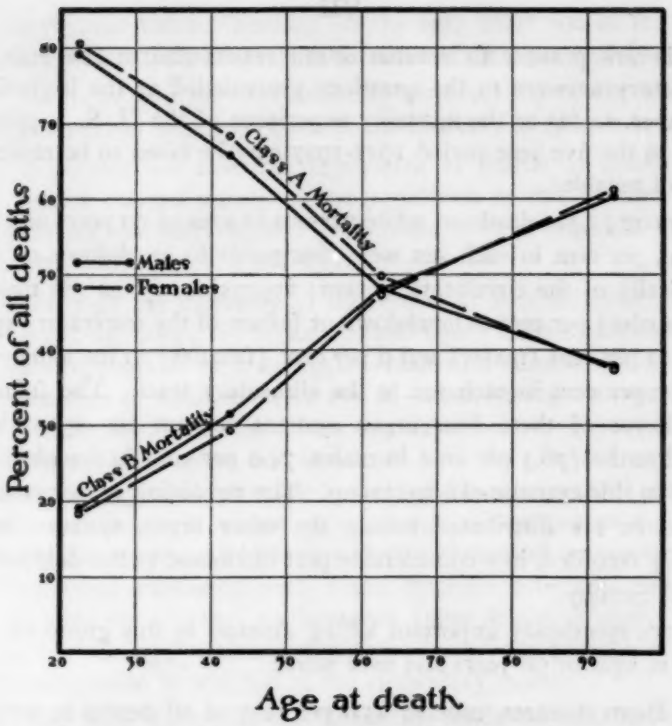

FIG. 9. THE TRENDS, AT FOUR ADULT AGE PERIODS FROM AGE 20-24 ON TO THE END OF THE LIFE SPAN, OF MORTALITY HAVING ITS PATHOLOGICAL LESIONS AND/OR CLINICAL MANIFESTATIONS ASSOCIATED WITH (A) ORGAN SYSTEMS NORMALLY IN DIRECT CONTACT WITH THE EXTERNAL ENVIRONMENT (PLUS EXTERNAL CAUSES EXCEPT SUICIDE); AND (B) ORGAN SYSTEMS NORMALLY NOT IN DIRECT CONTACT WITH THE EXTERNAL ENVIRONMENT (PLUS "SENILITY")

directly exposed to and in contact with the external environment (or due to direct lethal traumatic impacts), and the correlative *increase* with advancing adult age of the percentage of mortality associated with organ systems not exposed to direct contact with the external environment (plus deaths recorded as due to "Senility") could not be more clearly apparent.

VII

It is now possible to summarize our results and to give reasonably satisfactory answers to the questions propounded at the beginning of this paper, so far as the mortality experience of the U. S. Registration States in the five year period 1923-1927 may be taken to be representative and reliable.

Among 72,320 deaths of white persons at ages of 90 years and above, over 45 per cent in each sex were chargeable to breakdown or failure biologically of the circulatory system; approximately 12 (in males) to 13 (females) per cent to breakdown or failure of the respiratory system; about 12 per cent (males) and 9 per cent (females) to the kidneys; and about 7 per cent in each sex to the alimentary tract. The failures or breakdowns of these four organ systems account for approximately three-fourths (76.3 per cent in males, 74.0 per cent in females) of all deaths in this extreme old-age group. The remaining 25 per cent of all the deaths are distributed among the other organ systems, and, as officially recorded, in a considerable part attributed to the dubious cause rubric "Senility."

More specifically important killing *diseases* in this group of whites dying at ages of 90 years and over were:

1. Heart diseases, causing 23.6 per cent of all deaths in males and 23.5 per cent in females.
2. Diseases of the arteries, causing 10.6 per cent of all deaths in males and 9.5 per cent in females.
3. Chronic nephritis, causing 10.6 per cent of all deaths in males and 8.2 per cent in females.
4. Cerebral hemorrhage causing 9.8 per cent of all deaths in males and 10.6 per cent in females.
5. External causes (except suicide), causing 4.3 per cent of all deaths in males and 6.5 per cent in females.
6. Bronchopneumonia, causing 4.0 per cent of all deaths in males and 4.7 per cent in females.
7. Lobar pneumonia, causing 2.6 per cent of all deaths in males and 2.5 per cent in females.

The dubious rubric "Senility" is the only other one of importance, with 12.8 per cent of all deaths for males and 14.0 per cent for females.

From these data the overwhelming responsibility of the circulatory system in setting the upper limit of the human life span is apparent.

It is pointed out that the registration of deaths by causes was so poor for the colored moiety of the population in this extreme old age group during the years studied as to make it quite impossible to reach any trustworthy comparative results.

A study of the mortality of the whites at four different adult age periods (20-24, 40-44, 60-64, and 90 and over) leads to the observation that there appears to be a steady *decrease* with advancing adult age of the percentage of total mortality due to causes having their pathological lesions and clinical manifestations associated with organ systems normally directly exposed to and in contact with the external environment (or due to direct lethal traumatic impacts of the environment); and a correlative *increase* with advancing adult age of the percentage of total mortality due to causes which have their pathological lesions and clinical manifestations associated with organ systems not exposed to direct contact with the external environment (plus deaths officially recorded as due to "senility," which in the 90 and over age group are probably chiefly assignable in real fact to the circulatory system).

It has been shown in the preceding study in this series [32] that longevity itself behaves as an inherited character. Parents and grandparents are differentiated in respect of their own duration of life in accordance with the ages at death of their children, and conversely children of long-lived parents live longer, on the average, than children of short-lived parents. What is really inherited and finds its expression in the results which have been stated regarding duration of life in years, is presumably the organic or biological *constitution* of the body. The present study perhaps throws some light on this by showing how the different organ systems stand up under the strain of long-living.

Nonagenarians are a selected lot of people. They are the ultimate survivors after all the rest of mankind has gone, unable to meet the vicissitudes of life and keep on living. The nonagenarians are such because they have organically superior constitutions, resistant to infections, soundly organized to function efficiently as a whole organism,

[32] Pearl, R. Studies on human longevity. IV. The inheritance of longevity. Preliminary report. HUMAN BIOLOGY, Vol. III, pp. 245-269, 1931.

and keep on doing it for a very long time. The observations on mortality indicate that throughout life infections and other harmful environmental forces are, on the whole, tending to take off the weaker and leave the stronger. Medical knowledge and skill, improved sanitation, and better conditions of life generally, have been, and are, able to prevent an increasingly larger amount of what may be called premature mortality, meaning that occurring before age 50, let us say. Especially have these agencies been able to reduce the lethal effects of infections, or at least to postpone to a later part of the life span their fatal action. But ultimately there is left a group of extremely old people, for whom on the whole infections have no particular terrors. They have been, and to a remarkable degree still are, able successfully to resist them. These people eventually die, to be sure. But a great part of them die, not because the noxious forces of the environment kill them, but because their vital machinery literally breaks down, and particularly that important part of it the circulatory system. To be sure the heart and arteries often have been injured by infections successfully weathered in earlier life which left their scars on organs thereafter in greater or less degree impaired. But, as the quotation from Humphry in the earlier part of this paper shows, many a nonagenarian has escaped even such damage.

Taking all the evidence into consideration it seems a fairly safe conclusion that if the upper limit of the human life span (as distinguished from the average duration of life [23]) is to be materially extended beyond what it now is it will be necessary for man to acquire, by some means not yet clear, a circulatory system organically superior to the already extremely good one—taken at its best—that he now has.

[23] For a detailed discussion of this distinction see Pearl, R., Span of life and average duration of life. *Natural History*, New York, Vol. 26, pp. 26-30, 1926.

NOTES

BIRTH RATE AND DENSITY IN A LOGISTIC POPULATION*

As Lotka[1] has shown, the births in a population growing according to the logistic law

$$N(t) = \frac{N_\infty}{1 + e^{-rt}} \tag{1}$$

may be expressed by the function

$$B(t) = \frac{N_\infty}{L_0}\left\{\phi(t') + \frac{\lambda_2}{2!}\phi''(t') + \ldots\right\} \tag{2}$$

where $\phi(t') = \frac{1}{1 + e^{-rt'}}$, and $t' = t + \lambda_1$. In other words, the fundamental component in the curve of births, which accounts for nearly the entire contingent of births, is itself a logistic curve. The birth rate per head is therefore closely approximated by

$$b(t) = \frac{1}{L_0}\frac{\varphi(t')}{\varphi(t)} \tag{3}$$

Since the area in which the population grows is constant, the density of population, $d(t)$, is proportional to $N(t)$.

$$d(t) = K\phi(t) = \frac{K}{1 + e^{-rt}} \tag{4}$$

where K is a proportionality constant.

Equations (3) and (4) express birth rate and density as functions of time. By eliminating t we can obtain the birth rate of a logistic population as a function of its density.

$$b(t) = \frac{K}{L_0}\frac{\varphi(t')}{d(t)} \tag{5}$$

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* From the Department of Biology of the School of Hygiene and Public Health of the Johns Hopkins University.

[1] Lotka, A. J. The structure of a growing population. HUMAN BIOLOGY, Vol. 3, pp. 459-493. December, 1931.

$$= \frac{K}{L_0 d(t)\ [1 + e^{-r(t+\lambda_1)}]} \tag{6}$$

But from equation (4)

$$t = -\frac{1}{r} log \frac{K-d}{d} \tag{7}$$

Substituting this in equation (6) we have

$$b = \frac{K}{L_0\, d(1 + \frac{K-d}{d}\, e^{-r\lambda_1})} \tag{8}$$

$$= \frac{K}{L_0[(1 - e^{-r\lambda_1})d + Ke^{-r\lambda_1}]} \tag{9}$$

$$= \frac{K'}{d + c} \tag{10}$$

where $K' = \frac{K}{L_0(1 - e^{-r\lambda_1})}$ and $c = \frac{Ke^{-r\lambda_1}}{1 - e^{-r\lambda_1}}$

But equation (10) is the functional relation between the mean free path of the molecules of a gas and its density for the case where the surface of the container is not negligible in comparison with the total area of the molecules.[2] Pearl [3] has found that the functional relation between egg production and density in experimental populations of Drosophila follows this law and presents evidence for a rational biological explanation of this result. Although the fact that the relation between reproductive rate and density in a logistic population follows the same functional form as the relation between mean free path and density in a gas does not prove that the underlying mechanisms are the same, it certainly suggests that they may be. This view is further supported by the experimental results of Pearl.

JOHN R. MINER.

[2] Loeb, L. B. Kinetic Theory of Gases. New York and London (McGraw-Hill Book Co., Inc.), 1927. Pp. xvi + 555.

[3] Pearl, R. The influence of density of population upon egg production in *Drosophila melanogaster*. [In press.]

QUOTATIONS

JOSEPH GLANVILL, A FORGOTTEN SEVENTEENTH CENTURY ICONOCLAST*

"A man cannot drink half the quantity of strong drinks in the bath that he can out of it; but if he hath drank before to excess, it allay's much, and is a great refreshment to the body. The bath provoketh urine." So wrote Joseph Glanvill in the fourth volume (p. 980) of the *Philosophical Transactions*[1] of the Royal Society. Such a man surely deserves to be resurrected from the oblivion to which he has been so carelessly consigned. But Glanvill, admirer of Montaigne, Bacon, and Descartes, and friend of Rous, Baxter, Boyle, and Casaubon, was also interested in things other than strong drinks and medicinal waters. He dedicated himself primarily to the destruction of the scientific and

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* Paper No. 164 from the Department of Biostatistics, School of Hygiene and Public Health, The Johns Hopkins University, Baltimore, Maryland.

[1] *Observations concerning the Bath-Springs communicated by Mr. Joseph Glanvill in a letter to the publisher dated June 16, 1669: at the Bath,* pp. 977-982. On page 981: "The Bath-guides live to a very great age, sometimes to near 100 years; ordinarily, if they are temperate, to 70. There are two at this time above 80, a man and his wife." And on the same page: "When women have washt their hair with the mixture of beaten eggs and oatmeal, this will poison the Bath so as to beget a most noisome smell casting a sea-green on the water which otherwise is very pure and limpid. This will taint the verry walls, and there is no cleansing of it but by drawing the Bath." In the second and third volumes of the same *Transactions,* pp. 525-527 and pp. 767-771, respectively, the publisher devoted a total of eight pages to Glanvill's *Answers to some of the inquiries formerly publish'd concerning mines.* These eight pages and the six just referred to contain all that Glanvill ever had published in the *Transactions.* In the third volume, page 771: "Concerning subterraneous Daemons, they have never seen any, but sometimes have heard knockings beyond their own works, which, when followed by them, have afforded plenty of ore. . . . . About 2 years since, one King of Wells in his Groove found a piece of Ore in which they fancied the shape of a Man, Eyes, Armes, Leggs, full Breast: The whole was about 4 inches in length; the Mine proved rich."

religious dogmatism[2] that prevailed in England during the first half of the seventeenth century. His mind revolted not only from the dogmatism which found all wisdom within the covers of Aristotle's works,[3] but also, no less from that form of dogmatism which flew to the opposite extreme.[4] Glanvill was a genuine *sceptic*.[5] According to him "sceptics seek truth in the book of nature, and in that search proceed with wariness and circumspection without too much forwardness in establishing maxims and positive doctrines; their opinions are proposed as hypoth-

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[2] See Heinrich Cornelius Agrippa von Nettesheim (1486-1535), *De Incertitudine et Vanitate Scientiarum et Artium atque Excellentia Verbi Dei Declamatio*, written in his forty-first year and published in Antwerp in 1531, for a sarcastic attack, first, on the existing sciences, second, on the pretensions of learned men, and, third, on the charlatanism of the period. A small octavo edition in English appeared in London in 1684.

About fifty years ago in 1881, Josiah Royce wrote in an essay, *The Decay of Earnestness:* "We need a more thorough-going, a more searching—yes, a more critical and skeptical—thought than any now current. For current thought is, in fact, naïf and dogmatic, accepting without criticism a whole army of ideas because they happen to be useful as bases for scientific work." *Fugitive Essays*, 8vo., Cambridge, [Mass.], 1920; p. 304.

[3] "Ancient thought, so far as it is positive, fails on account of its neglect to criticise its assumptions; so far as it is negative, it fails, because it forgets that proof of the inconsistencies of the terms in which we symbolize things has nothing to do with the cogency of the logic of facts. The negations of Pyrrhonism [Glanvill was unjustifiably made a disciple of Pyrrho by a writer of the last century who preferred to remain anonymous.] are as shallow, as the assumptions of Platonism are empty. Modern thought has by no means escaped from perversions of the same order. But, thanks to the sharp discipline of physical science, it is more and more freeing itself from them. In face of the incessant verification of deductive reasoning by experiment, Pyrrhonism has become ridiculous; in face of the ignominious fate which always befalls those who attempt to get at the secrets of nature, or the rules of conduct, by the high *a priori* road, Platonism and its modern progeny show themselves to be, at best, splendid follies." Huxley, *Hume: with helps to the study of Berkeley*, 8vo., London, 1894; p. ix.

[4] "When I say that Descartes consecrated doubt, you must remember that it was that sort of doubt which Goethe has called 'the active scepticism, whose whole aim is to conquer itself' and not that other sort which is born of flippancy and ignorance, and whose aim is only to perpetuate itself, as an excuse for idleness and indifference." Huxley, *Method and Results*, 8vo., London, 1893; p. 170.

[5] See Hermann Lotze, *Logic*, edited by B. Bosanquet, 2v., 8vo., Oxford, 2nd ed., 1887; v.2, bk. 3, ch.1, *On Scepticism*, pp. 176-199. Also Hume, *Treatise of Human Nature*, edited by L. A. Selby-Bigge, 8vo., Oxford, [1896]; bk. 1, pt. 4, *Of the Sceptical and other Systems of Philosophy*, pp. 180-274.

eses that may probably be the true accounts without peremptorily affirming that they are." [6]

Glanvill was born in Plymouth in 1636 and died forty-four years later at Bath in 1680. At Oxford he came under the influence of John Wilkins and Wallis, and one of his intimate friends was Francis Willoughby, who later became an authority on birds and fishes. In the same year, 1658, that he was made M. A. he became chaplain to Francis Rous, and the following year when Rous died he returned to Oxford. After holding livings in Essex and Somersetshire he was named rector of the abbey church at Bath where he resided until his death. Such are, in brief, the chronological dry bones of his life.

His first attempt to disturb the equilibrium of the age was made by his publishing in 1661, *The Vanity of Dogmatizing or Confidence in Opinions manifested in a Discourse of the Shortness and Uncertainty of our Knowledge, and its causes, with some Reflexions on Peripateticism, and an apology for Philosophy,* his first and his most original book.[7] After its appearance the author was immediately charged with scepticism [8] and atheism. Of the book Henry Hallam [9] said that it was a remarkable piece of writing but so scarce as to be hardly known at all except by name. This work was republished, considerably altered, by Glanvill in 1665 under the title *Scepsis Scientifica* [10] and is even rarer

[6] Quoted by John Owen, *Scepsis Scientifica,* 12mo., London, 1885; p. xviii.

[7] This work suggested the story of *The Scholar Gypsy* to Matthew Arnold.

[8] "We condemn as sceptics all who do not share our illusions, ..." Anatole France, *The Garden of Epicurus,* 8vo., London and New York, 2nd ed., 1920; p. 101.

[9] *Introduction to the Literature of Europe,* 4v., 8vo., London, 5th ed., 1855; v. 4, p. 119.

[10] See reproduced title-page (Figure 1).

Small 4to. A^{4-1}, (a)-$(c)^4$, $(d)^{4-2}$, B-Z^4, Aa^4, A^4, $(a)^4$, B-M^4, N^{4-2}. [34] + 184 + [16] + 92 pp.+ plate. 28 lines + headline and signature and catchword line, 16 (17.6) × 11.4 cm. Page size of present copy, 19.4 × 15.2 cm.

$A2^a$, title-page; $A2^b$, blank; $A3^a$, plate [Royal Society arms]; A3, A4, (a)-$(c)^4$, address to the Royal Society; $(d)1^a$, *imprimatur* dated Octob. 18, 1664; $(d)1^b$, blank; $[(d)2]^a$, page of errata; $[(d)2]^b$, blank; $B1^a$(p. 1)-$Z2^b$(p. 172), text of *Scepsis Scientifica;* $Z3^a$(p. 173)-$Aa4^b$(p. 184), text of *An Apology for Philosophy;* $A1^a$, title-page of second pagination: *Scir* $\frac{E}{I}$ *tuum nihil est: / OR, /* The AUTHORS *DEFENCE* / OF / The Vanity of DOGMATIZING; / Against the / EXCEPTIONS / OF / The Learned *THO. ALBIUS* /In his Late / SCIRI. / [single rule] / No doubt *but* ye *are the Men, and Wisdom shall dye / with you!* Job. / [single rule] / *LONDON,* / Printed by *E. C.* for *Henry Eversden* at the Grey-Hound / in St. *Pauls*-Church-Yard, 1665. ; $A1^b$, blank; $A2^a$-$A3^a$, *The Authors Apology for his Style;* $A3^b$, blank; A4, $(a)^4$, *To the Learned Tho. Albius;* $B1^a$(p. 1)-$L2^a$(p. 75), text of *The Authors Defence of the Vanity of Dogmatizing;* $L2^b$, blank; $L3^a$(p. 77)-$N2^b$(p. 92), text of *A Letter to a Friend concerning Aristotle.*

than its predecessor, a large part of the edition having been destroyed in the fire of 1666.

The *Scepsis* has been chosen for quotation. Because of its extreme rarity [11] the following brief synopsis of the work has been prepared. This will enable the reader to determine the probable genesis of the material that will be quoted presently and its relationship to other parts of the work. Glanvill begins with a toothsome, eulogistic dedication to the Royal Society. This panegyric was at that time sufficiently discriminating to obtain for him the F. R. S. The first three chapters deal with primitive ignorance, decay of intellect, and ignorance of self. Then follow five chapters (4-8) that introduce subjects about which little is known: the soul, the body, and the motion of a wheel. He turns his attention next, in nine chapters (9-17), to six causes of human ignorance: superficiality, limitations of the senses, fallacies of the senses, fallacies of the imagination, assent to slightly examined conclusions, and affections that lead to unwarrantable conclusions. The five chapters (18-22) that follow are devoted to reflections, under six headings, against Aristotelian philosophy: verbosity, litigiousness, absurd hypotheses and definitions, barrenness, inconsistencies with divinity, and self-contradictoriness. Three chapters (23-25) question whether there is any science in the sense of the dogmatists: the superficiality of the dogmatists, they draw conclusions in a narrow domain and make universal application, they rashly conclude three things [12] impossible which are likely to be otherwise, and, they imply that knowledge is possible without knowledge of first causes and that some causes may be known without knowing all causes. The next chapter (26) inquires into the infallibility of the senses. And the final chapter (27) gives six reasons why dogmatism should not survive: dogmatism is the effect of ignorance, it dwells with untamed passions and ungoverned spirit, it is the great disturber of the world and its people, it is ill manners and immodesty, it holds men captive in error, and it betrays a poverty and narrowness of spirit.

The title-page of the *Scepsis* is reproduced and appears as Figure 1.

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[11] Owen's reprint of 1885 is quite unobtainable. The second pagination, [16] + 92, and the plate are omitted.

[12] *First*, the power of one man's spirit over that of another, *second*, momentous conveyance at almost any distance, and, *third*, the sympathetic cure of wounds. In connection with the last, see Sir Kenelm Digby, *Of Bodies, and of Man's Soul, to discover the Immortality of Reasonable Souls*, 4to., London, 1669; *Of the Powder of Sympathy*, pp. 145-205 (second pagination).

Scepsis Scientifica:

OR,

Confest *Ignorance*, the way to *Science*;

In an *Essay* of

The Vanity of DOGMATIZING,

AND

CONFIDENT Opinion.

WITH

A REPLY to the EXCEPTIONS

Of the Learned

THOMAS ALBIUS.

By JOSEPH GLANVILL, *M. A.*

LONDON:

Printed by *E. Cotes*, for HENRY EVERSDEN at the *Gray-Hound* in *St. Paul's Church-yard.* M. DC. LXV.

FIG. 1. TITLE-PAGE OF SCEPSIS SCIENTIFICA, 1665.
(The Original measures 18.2 × 12.2 cm.)

One could with very little difficulty find many paragraphs of uncommon vivacity and acuteness in this forgotten tome that would lend themselves admirably for quotation. Chapters 15, 16, and 17, where Glanvill analyzes the sixth cause of ignorance, have been selected. This cause, which is *affections that lead to unwarrantable conclusions,* is considered under five subheads: natural disposition, prejudices of custom and education, personal interest, man's love of his own productions, and reverence for antiquity and authority.

CHAPTER 15. 1. *Congruity* of Opinions, whether true or false, to our *natural constitution,* is one great incentive to their reception; for in a sense the *complexion* of the *mind,* as well as *manners,* follows the *Temperament* of the Body. On this account some men are genially disposed to some *Opinions,* and naturally as averse to others. And we *love* and *hate* without a known cause of either. Some Faces both of Persons and Things, we admire and dote on: others, in our impartial apprehensions no less deserving our esteem, we cannot behold without resentment; yea it may be with an invincible disregard. And I question not, but *intellectual* representations are received by us, with as unequal a Fate upon a bare *Temperamental* Relish or Disgust: the *Understanding* also hath its *Idiosyncrasies,* as well as other faculties. So that the great stirrs of the disputing World, are but the conflicts of the humours. *Superstition, Atheism,* and *Enthusiasm*[12] are tempers; not mere infusions of *Education,* and *Opinion.* Indeed the dull and unactive spirits that concern not themselves in *Theory,* follow the swinge of the common belief in which they were first instructed: but the more *vigorous* and *stirring* will fall into *that* of their particular *Crasis.* And when the humour is awakened, all the bonds of Custome and Education cannot hold them. The opinions which are suited to their respective tempers will make way to their assent, in spight of accidental preingagements. Thus *opinions* have their *Climes* and *National* diversities: and as some regions have their proper Vices, not so generally found in others; so have they their mental depravities, which are drawn in with the air of their Countrey. And perhaps this is a considerable cause of the diversity of *Lawes, Customes, Religions, natural* and *moral* Doctrines, which is to be found in the divided Regions of the inhabited Earth. . . . .

And if we do but more nearly look into our *faculties,* beginning our survey from the lowest dregs of *sense,* even those which have a nearer commerce with *matter,* and so by steps ascend to our more *spiritualiz'd selves*: we shall thoroughly discover how *constitutional partiality* swayes us. To begin then at the *Sences;* that to one *Palate* is *sweet,* and *delicious,* which to another, is *odious* and *distastful;* or more compendiously in the Proverb, *One man's meat is another's poyson.* What to one is a most grateful *odour,* to another is *noxious* and *displeasant;* and 'twere a misery to some to lye stretch't on a bed of Roses: that's a *welcome touch* to one, which is *disagreeing* to another; the same *Aires* which some entertain with most delightful transports, to others are importune; and the objects which *this* man can't *see* without an *Extasie, that* is no more mov'd at than a *Statue.* Now the *phancies*

[12] For Glanvill's meaning of *enthusiasm,* see Locke, *Essay concerning Human Understanding,* edited by A. C. Fraser, 2 v., 8vo., Oxford, 1894; v. 2, ch. 19.

of the most, like the *Index* of a Clock, are moved but by the inward *Springs* and *Wheels* of the corporal *Machine;* which even on the most sublimed Intellectuals is dangerously *influential.* And yet this sits at the Helm of the Worlds belief; and Vulgar *Reason* is no better then a more *refined imagination.* So then the *Senses, Phancy,* and what we call *Reason* it self, being thus influenced by the *Bodies temperament,* and little better then indications of it; it cannot be otherwise, but that this *Love of our selves* should strongly incline us in our most *Abstracted Dijudications.*

CHAPTER 16. 2. Another branch of this *selfish fondness,* by reason of which we miscarry of *Science,* is the almost insuperable *prejudice* of *Custom,* and *Education*: by which our minds are encumber'd, and the most are held in a *Fatal Ignorance.* Yea could a man be composed to such an advantage of constitution, that it should not at all adulterate the images of his mind; yet this *second nature* would alter the *crasis* of the Understanding, and render it as obnoxious to aberrances, as now. Thus we judge all things by our *anticipations;* and condemn or applaud them, as they agree or differ from our *first receptions.* One countrey laughs at the *Lawes, Customs,* and *Opinions* of another, as absurd and ridiculous; and the other is as charitable to them, in its conceit of theirs. . . . . There is nothing so absurd, to which *education* cannot form our ductile *minority;* it can lick us into shapes beyond the *monstrosities of Africa.* And as King *James* would say of *Parliaments, It can do any thing but make a man a woman.* For our initial age is like the melted wax to the prepared Seal, capable of any impression from the documents of our Teachers. We seldom examine our Receptions more than children do their *Catechisms;* but by a *careless greediness* swallow all at a venture. For *Implicit* faith is a vertue, where *Orthodoxie* is the object. We are bound to our Countreys *Opinions,* as to its *Laws*: and an accustomed assent is *tantamount* to an infallible conclusion. He that offers to dissent, shall be an *Out-law* in reputation: and the fears of guilty *Cain,* shall be fulfilled on him, who ever *meets* him *shall slay him.* Thus *Custom* and *Education* have sealed the *Canon;* and he that adds or takes away from the Book of *Orthodox* belief, shall be more than in danger of an *Anathema*: And the *Inquisition* is not confined to the jurisdiction of the *Triple-Crown.* The rankest follies are *Sacred,* if *customary;* and the *Fashion* is *handsome,* and *agreeable,* though never so *uncouth* to an unconcerned beholder. And as we are fond of every thing with which *custom* hath acquainted us; so on the other hand we start and boggle at every *unusual* appearance, and cannot endure the sight of the *bug-bear, Novelty.* On this account very innocent truths are often affix't with the reproach of *Heresie;* and made terrible things in the imaginations of their misinform'd and frighted enemies; who like children scared in the dark, fly the *Monsters* of their *Phancies,* and dare not stay to take a true account of the object of their fears. So that there is scarce any truth, but its adversaries have made it an ugly *Vizard;* by which it's exposed to the hate and disesteem of superficial examiners: For an opprobrious title with vulgar believers is as good as an *Argument.* And 'tis but writing the name that customary receptions have discredited, under the opinions we dislike; and all other refutation is superfluous. Thus shallow apprehenders are frighted from many sober *Verities;* like the king of the *Arabs,* who ran away from the *smoaking Mince-Py,* apprehending some dangerous plot in the harmless steam.

So then, while we thus mistake the infusions of *education*, for the *principles* of universal *nature;* we must needs fail of a *scientifical Theory*. And therefore the two Nations differing about the *antiquity* of their language, made appeal to an undecisive *experiment;* when they agreed upon the tryal of a child brought up among the wild inhabitants of the Desert. The *Language* it spake, had no reason to be accounted the most ancient and natural: And the lucky determination for the *Phygians* by its pronouncing the word *Beck*, which signified Bread in the dialect of that Countrey, they owed not to *Nature*, but the *Goat-herd;* from which the exposed infant, by accompanying that sort of *animals*, had learnt it.

3. *Interest* is another thing, by the *magnetisme* of which our *affections* are almost irresistibly attracted. It is the *Pole* to which we turn and our *sympathizing* Judgements seldom decline from the *direction* of this *Impregnant*. Where *Interest* hath engaged men; they'l find a way to Truth, or make one. Any thing is *good* and *true*, to one whose *Interest* it is, to have it so. And therefore Self-designers are seldome disappointed, for want of the speciousness of a cause to warrant them; in the belief of which, they do oft as really impose upon themselves, as they industriously endeavour it upon others. With what an infinite of *Law-suits, controversies*, and *litigious cases* doth the world abound? and yet every man is confident of the truth and goodness of his own. And it may be as Master *Hobbs* observes, one reason that Mathematical demonstrations are uncontroverted, is, because *Interest* hath no place in those unquestionable *verities*: when as, did the advantage of any stand against them, perhaps *Euclid's Elements* would not pass so universal a suffrage. . . . .

4. Besides, fourthly, *Self-love* engageth us for any thing, that is a *Minerva* of our own. And thereby detains us in the snares of *ignorance* and *folly*. We love the issues of our *Brains*, no less than those of our *bodies*: and fondness of our own *begotten notions*, though *illegitimate*, obligeth us to maintain them. We hugge intellectual deformities, if they bear our Names; and will hardly be perswaded they are so, when our selves are their Authors. If their *Dam* may be judge, the young *Apes* are the most beautiful things in Nature; and if we might determine it, our proper conceptions would be all voted *Axioms*.

So that to give the sum of all, most of the contests of the litigious world pretending for *Truth*, are but the bandyings of one man's *affections* against another's: in which, though their reasons may be foil'd, yet their *Passions* lose no ground, but rather improve by the *Antiperistasis* of an opposition.

CHAPTER 17. 5. Another thing, that engageth our *affections* to unwarrantable conclusions, and is therefore fatal to *Science;* is our doting on *Antiquity*, and the opinion of our fathers.[14] We look with a superstitious reverence upon the accounts of praeterlapsed ages: and with a supercilious severity, on the more deserving products of our own. A vanity, which hath possess'd all times as well as ours; and the

[14] *Cf.* Hobbes, *Leviathan*, edited by A. R. Waller, 8vo., Cambridge, [England], 1904, p. 23: " . . . . he that takes up conclusions on the trust of Authors, and doth not fetch them from the first Items in every Reckoning, (which are the significations of names settled by definitions), loses his labour; and does not know any thing; but onely beleeveth."

Also Huxley, *Method and Results*, p. 40: "The improver of natural knowledge absolutely refuses to acknowledge authority, as such. For him scepticism is the highest of duties; blind faith the one unpardonable sin."

golden Age was never *present.*[25] For as in *Statick* experiment, an inconsiderable weight by virtue of its distance from the Centre of the Ballance, will preponderate much greater magnitudes; so the most slight and chaffy opinion, if at a greater remove from the present age, contracts such an esteem and veneration, that it outweighs what is infinitely more ponderous and rational, of a *modern* date. We reverence gray-headed Doctrines; though feeble, decrepit, and within a step of dust: and on this account maintain opinions, which have nothing but our *charity* to uphold them. While the *beauty* of a Truth, as of a *picture* is not acknowledg'd but at a *distance;* and that wisdom is nothing worth, which is not fetcht from *afar:* wherein yet we oft deceive our selves, as did that *Mariner,* who mistaking them for precious stones, brought home his ship fraught with common *Pebbles* from the remotest *Indies.* Thus our Eyes, like the *preposterous Animals,* are behind us; and our intellectual motions *retrograde.* We adhere to the determinations of our fathers, as if their *opinions* were entail'd on us as their *lands;* or (as some conceive) part of the Parents soul were portion'd out to his off-spring, and the conceptions of our minds were *ex traduce.* The Sages of old live again in us; and in opinions there is a *Metempsychosis.* We are our re-animated *Ancestours,* and antedate their *Resurrection.*

And thus, while every age is but another shew of the former; 'tis no wonder, that Science hath not out-grown the dwarfishness of its *pristine stature,* and that the *Intellectual world* is such a *Microcosm.* . . . .

Now if we enquire the reason, why the *Mathematicks,* and *Mechanick Arts,* have so much got the start in growth of other *Sciences*: we shall find it probably resolv'd into this, as one considerable cause: that their progress hath not been retarded by that reverential awe of former discoveries, which hath been so great an hinderance to Theoretical improvements. 'Twas never an heresie to out-limn *Apelles;* nor criminal to out-work the *Obelisks. Galilaeus* without a crime outsaw all *Antiquity,* and was not afraid to believe his eyes, in spight of the *Opticks* of *Ptolomy* and *Aristotle.* 'Tis no discredit to the *Telescope* that antiquity ne're saw in't: Nor are we shy of assent to those *celestial* informations, because they were *hid from ages.* We believe the *verticity* of the *Needle,* without a Certificate from the *dayes of old*: And confine not our selves to the sole conduct of the *Stars,* for fear of being wiser then our Fathers.

'Twas this vain Idolizing of Authors, which gave birth to that silly vanity of *impertinent citations;* and inducing *Authority* in things neither requiring, nor deserving it. That saying was much more observable, *That men have beards and women none;* because quoted from Beza. But these ridiculous fooleries, signifie nothing to the more generous discerners, but the *Pedantry* of the affected *Sciolist.* . . . . 'Tis better to own a Judgment, then a *memory* like a Sepulchre furnished

[25] *Cf.* Royce, *loc. cit.*, p. 300: "Every active period of history thinks its activity of prodigious importance, and its advance beyond its predecessors very admirable. . . . . Like individuals, then, whole eras of history go by, sublimely confident in their own significance, yet often unable to make their claims even interesting in the sight of posterity."

Also Havelock Ellis, *The Dance of Life,* 12mo., New York, reprint of 1929, p. 272: "It gratifies us to imagine that, as a race, we have reached a point on the road of progress beyond that vouchsafed to our benighted predecessors, and that, as individuals or as nations, it is given to us, fortunately—or rather, through our superior merits—to enjoy a finer degree of civilization than the individuals and the nations around us."

with a load of broken and discarnate bones. *Authorities* alone with me make no number, unless Evidence of Reason stand before them: For all the *Cyphers* of *Arithmatick*, are no better then a single *nothing*. And yet this rank folly of affecting such impertinencies, hath overgrown our Times; and those that are Candidates for the repute of *Scholars*, take this way to compass it. But again, to what is more perpendicular to our discourse, if we impartially look into the remains of *Antique* ages; we shall find but little to justifie so groundless a Tyranny, as *Antiquity* hath imposed on the enslaved world. For if we take an account of the state of *Science*, beginning as high as History can carry us; we shall find it still to have lain under such unhappy disadvantages, as have hindered its advance in any considerable degrees of improvement. . . . . Though those former dayes have not wanted brave *Wits*, that have gallantly attempted, and made Essays worthy immortality; yet by reason either of the unqualified capacities of the multitude, (who dote on things slight and trivial, neglecting what is more rare and excellent) or the clamorous assaults of envious and more popular opposers, they have submitted to Fate, and are almost lost in *Oblivion*. And therefore, as that great man, the *Lord Bacon* hath observed, *Time* as a *River*, hath brought down to us what is more light and superficial; while things more solid and substantial have been immersed.

WILLIAM M. GAFAFER

RECENT LITERATURE

USEFUL IN THE STUDY OF HUMAN BIOLOGY

In this department will be noticed the books and reprints which are currently received at the Editorial Office. Publications intended for notice here should be addressed to Dr. Raymond Pearl, Department of Biology, School of Hygiene and Public Health, 1901 East Madison Street, Baltimore, Maryland, U. S. A.

AN EXPLANATORY NOTE REGARDING A MORE DETAILED CLASSIFICATION OF RECENT LITERATURE USEFUL IN THE STUDY OF HUMAN BIOLOGY

BY RAYMOND PEARL

After three years experience it seems likely that the usefulness of this department of HUMAN BIOLOGY will be enhanced by expanding in greater detail the classification under which the titles of books and memoirs are here presented. It has been the aim of the editors of this journal from the beginning to make this department of real value to workers in the field. To this end a great deal of painstaking care has gone into its compilation and editing, particularly to ensure (subject of course to our human liability to err) that the bibliographic description of the items included shall be sufficiently detailed in the first place, precise and accurate in the second place, and, finally, indicative (by means of notation [Bibliography of ..... titles]) of additional bibliographic sources.

The broad classification under eight comprehensive headings with which the listing started, and which has been maintained since, was based primarily upon purely pragmatic and utilitarian considerations. This is also true of the expansion into more detailed headings upon which we are now embarking. Attempts at completely logical classification of the content of the sciences or of any particular science have never achieved entire success. Or perhaps it is better to say, have never fully satisfied anyone except those who made them. The principal reason why this is so appears to be that scientific workers do not limit either the nature of their ideas or the exposition of them to the boundaries set by the walls of the pigeon holes in any classificatory scheme, however nobly conceived. Every day, with the advancement of knowledge, the necessary interrelationship and interdependence of the various sciences becomes more evident. As knowledge increases so also does our perception of the intricate and integrated complexity of Nature. But in the practical conduct of life classifications are pragmatically essential. They help rather than hinder, though they may be logically imperfect.

Beginning with this issue recent literature will be classified under the following heads:

I. EVOLUTION AND GENETICS
1. *Evolution—General Principles*
2. *Human Evolution*
3. *Prehistory and Archeology*
4. *Genetics—General Principles*
5. *Human Heredity*
6. *Eugenics*

II. GROUP BIOLOGY
1. *Environment of Man*
2. *Public Health and Hygiene*
3. *Migration*
4. *Population*
5. *Mortality and General Vital Statistics*
6. *Morbidity and Epidemiology*
7. *Natality, Fecundity, Fertility*
8. *Birth Control*
9. *Marriage and Divorce*

III. BEHAVIOR
1. *Racial Psychology*
2. *Individual Psychology*
3. *Psychiatry*
4. *History and Biography*
5. *Sociology, Law, Politics, and Religion*
6. *Economics*
7. *Education*
8. *Ethnology, Cultural Anthropology, Travel and Exploration*

IV. SOMATOLOGY AND CONSTITUTION
1. *Anatomy*
2. *Physical Anthropology and Anthropometry*
3. *Constitution*

V. PHYSIOLOGY AND BIOCHEMISTRY
1. *Physiology*
2. *Senescence, Senility and Longevity*
3. *Biochemistry*
4. *Pharmacology*

VI. PATHOLOGY, CLINICAL MEDICINE AND SURGERY
(Not further divided for the present.)

VII. METHODOLOGY
1. *Mensurational and Observational*
2. *Mathematical and Statistical*
3. *Other and General*

VIII. GENERAL AND MISCELLANEOUS
(Not further divided.)

There appears to be no need for any extended explanatory discussion of this classification. Its logical imperfections and incongruities are apparent. But in actual practice we have found it workable and useful in putting the items which go over the editorial desk into pigeon holes. This is all that it is intended to accomplish.

Under heading I are put together those subjects which bear most directly upon the evolution of man, with the exception that the literature of *variation* in man, the correlative of heredity in the science of genetics, is placed under heading IV, because realistically it happens that the great majority of workers whose chief interest is in human heredity are different persons, and work in a different way and by mainly other techniques, than those whose main interest is in human types and variabilities (the physical anthropologists). Subheadings I. 3 (*Prehistory*, etc.), and I. 6 (*Eugenics*) might perhaps from some points of view quite as logically have been placed under heading III. But on the whole it seems better to put them in close relation to evolution and genetics respectively.

"Behavior" as heading III is not used in the sense of "behaviorism," but as a simple general designation of the activities of men.

Heading IV obviously might be further sub-divided than it is, and possibly will be later.

Sub-heading V. 2 (*Senescence*, etc.) is plainly a much more special topic than the other broad fields under the general heading. There is no logical excuse whatever for this. It is merely an expression of the writer's personal interest and the research activities of his laboratory.

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LeBuffe, Francis P. But if it be a fossil! *America*, Vol. XLVI, pp. 158-159, 1931.

Linton, Edwin. Adventures in evolution. *School and Society*, Vol. 34, pp. 76-83, 1931.

Price, George McC. The Geological-Ages Hoax. A Plea for Logic in Theoretical Geology. *New York* (Fleming H. Revell Co.), 1931. Pp. 126. 5 × 7½ inches. $1.25.

Rignano, Eugenio. The concept of purpose in biology. *Mind*, Vol. XL, N.S., pp. 335-340, 1931.

2. *Human Evolution*

Abel, Othenio. Die Stellung des Menschen im Rahmen der Wirbeltiere. *Jena* (Gustav Fischer), 1931. Pp. xvi + 398. 6¾ × 10⅛ inches. 20 marks (paper); 22 marks (cloth).

Bennett, Wendell C. Archaeology of Kauai. Bernice P. Bishop Museum Bulletin 80. *Honolulu* (Bernice P. Bishop Museum), 1931. Pp. iii + 156 + 15 plates. 6¾ × 10 inches. $2.00 (paper). [Bibliography of 55 titles.]

Bentley, Madison. In Quest of Glacial Man. A Plan of Coöperation between Excavators and the Representatives of the Sciences of Man and of the Earth. Prepared for National Research Council, Division of Anthropology and Psychology. *Reprint and Circular Series of the National Research Council* No. 100. *Washington* (National Research Council), 1931. Pp. 20. 6⅝ × 9⅛ inches. 40 cents (paper).

Binet-Sanglé, Dr. Les Ancêtres de l'Homme. *Paris* (Albin Michel), 1931. Pp. xx + 290. 5½ × 8½ inches. 25 francs (paper). [Bibliography of 11½ pages.]

Conklin, Edwin G. Science and the future of man. Address at the Graduate Convocation, Brown University, June 14, 1930. *Brown University Papers* IX. *Providence, R. I.* (Brown University), 1930. Pp. 16. 6 × 9 inches.

Kidder, A. V. The future of man in the light of his past: The view-point of an archaeologist. *Scientific Monthly*, Vol. XXXII, pp. 289-293, 1931.

Spiller, G. The Origin and Nature of Man. An Enquiry into Fundamentals. Reconciling Man's Proud Achievements with Man's Humble Descent. *London* (Williams and Norgate, Ltd.), 1931. Pp. xiv + 383. 6 × 9⅝ inches. 15 shillings net.

3. *Prehistory and Archeology*

Bartlett, Katherine. Prehistoric Pueblo foods. *Museum Notes, Museum of Northern Arizona* (*Flagstaff, Aris.*), Vol. 4, No. 4. Pp. 1-4, 1931. [Bibliography of 9 titles.]

Davis, Emily C. Ancient Americans. The Archaeological Story of Two Continents. *New York* (Henry Holt and Co.), 1931. Pp. xii + 311. 5¼ × 8 inches. $3.50. [138 references to excavations and publications.]

Murray, Louise W., Edited by. Selected Manuscripts of General John S. Clark Relating to the Aboriginal History of the Susquehanna. *69 S. Franklin St., Wilkes-Barre, Pa.* (Society for Pennsylvania Archaeology, Frances Dorrance, Secy.), 1931. Pp. xvi + 150. 6¾ × 10 inches. $2.00 (paper); $2.50 (cloth). [Bibliography of 61 titles.]

ROBERTS, FRANK H. H., JR. The ruins at Kiatuthlanna, Eastern Arizona. *Smithsonian Institution, Bureau of American Ethnology*, Bulletin 100. *Washington* (U. S. Government Printing Office), 1931. Pp. viii + 195. 5¾ × 9 inches. 65 cents (paper). [Bibliography of 60 titles.]

ROSS, SIR E. DENISON, Edited by. The Art of Egypt Through the Ages. *London* (The Studio, Ltd.); *New York* (William Edwin Rudge, Publisher), 1931. Pp. 354. 8¾ × 11¼ inches. 30 shillings (England); $15.00 (New York).

4. *Genetics—General Principles*

BLAKESLEE, ALBERT F. The genetic view-point. *Science*, Vol. 73, pp. 571-577, 1931.

FORD, E. B. Mendelism and Evolution. The Dial Press Monographs on Biological Subjects. *New York* (The Dial Press), 1931. Pp. xii + 116. 4 × 6½ inches. $1.50 [Bibliography of 86 titles.]

JOLLOS, VICTOR. Genetik und Evolutionsproblem. *Leipzig* (Akademische Verlagsgesellschaft m.b.H.), 1931. Pp. 44. 6 × 9 inches. 3 marks (paper).

5. *Human Heredity*

BLAKESLEE, A. F., and M. R. SALMON. Odor and taste blindness. *Eugenical News*, Vol. XVI, pp. 105-108, 1931.

BONNEVIE, KRISTINE. Was lehrt die Embryologie der Papillarmuster über ihre Bedeutung als Rassen- und Familiencharakter? Teil III. Zur Genetik des quantitativen Wertes der Papillarmuster. *Zeitschrift für induktive Abstammungs- und Vererbungslehre*, Bd. LIX, pp. 1-60 + 4 tables, 1931. [Bibliography of 20 titles.]

CREW, F. A. E., and WM. C. MILLER. Human sterility. A study of an unusual pedigree. *Eugenics Review*, Vol. XXIII, pp. 127-128, 1931.

EAST, E. M. The inheritance of mental characteristics. *Mental Hygiene*, Vol. XV, pp. 45-51, 1931.

FRETS, G. P. Eenige Voorbeelden van Erfelijkheid bij den Mensch. *Ned. Maandschrift voor Geneeskunde*, 1931, pp. 725-735.

FRETS, G. P. Über die Dominanz des brachycephalen Kopfindex. *Zeitschrift für Morphologie und Anthropologie*, Band XXIX, pp. 512-517, 1931.

GESELL, ARNOLD. The developmental psychology of twins. Chapter 6 of "A Handbook of Child Psychology," *Worcester* (Clark University Press), 1931. Pp. 158-203. [Bibliography of 107 titles.]

KOCH, HANS, and FRIDTJOF MJÖEN. Die Erblichkeit der Musikalität II. Geneostatistische Untersuchungen über die Erblichkeitsverhältnisse der Musikalität. *Zeitschrift für Psychologie*, Bd. 121, pp. 104-136, 1929.

MJØEN, JON ALFRED. Geniet og forbryderen som biologisk problem. I. Den store begavelse og dens arvegang. II. Forbryterske tendenser og deres arvegang. *Religion och Kultur*, Årgång II, pp. (of reprint) 1-30, 1931. [English summary, "Inheritance of ability," of 2 pages.]

MJØEN, JON ALFRED. Race-crossing and glands. Some human hybrids and their parent stock. *Eugenics Review*, Vol. XXIII, pp. 31-40, 1931. [Bibliography of 20 titles.]

SCHIEFFELIN, BARBARA, and GLADYS C. SCHWESINGER. Mental Tests and Heredity, Including a Survey of Non-Verbal Tests. *Cold Spring Harbor, L. I., N. Y.* (Eugenics Research Assoc.), 1930. Pp. ix + 298. 6 × 9⅛ inches. 75 cents (paper); $1.00 (cloth); + 15 cents postage. [Bibliography of 39 titles.]

STEGGERDA, MORRIS. A family of mechanics. *Eugenical News*, Vol. XVI, pp. 58-59, 1931.

STEGGERDA MORRIS. The inheritance of bronchial trouble. *Eugenical News*, Vol. XVI, pp. 26-28, 1931.

STEGGERDA, MORRIS. The inheritance of hair form. *Eugenical News*, Vol. XVI, p. 56, 1931.

STEGGERDA, MORRIS. Lack of scholastic interest. *Eugenical News*, Vol. XVI, p. 9, 1931.

WIENER, ALEXANDER S. Chances of detecting interchange of infants, with special reference to blood groups. *Zeitschrift für induktive Abstammungs- und Vererbungslehre*, Bd. LIX, pp. 227-235, 1931. [Bibliography of 12 titles.]

WIENER, ALEXANDER S. Determination of paternity by blood groups. *American Journal of the Medical Sciences*, Vol. CLXXXI, pp. 605-608, 1931. [Bibliography of 12 titles.]

WIENER, ALEXANDER S. Heredity of the agglutinogens M and N of Landsteiner and Levine. II. Theoretico-statistical considerations. *Journal of Immunology*, Vol. XXI, pp. 157-170, 1931. [Bibliography of 10 titles.]

6. *Eugenics*

EAST, E. M. The future of man in the light of his past: The view-point of a geneticist. *Scientific Monthly*, Vol. XXXII, pp. 301-308, 1931.

INTERNATIONAL FEDERATION OF EUGENIC ORGANIZATIONS. Report of the ninth conference of the International Federation of Eugenic Organizations, Farnham, Dorset, September 11th to 15th, 1930. *406 Fulham Road, London* (International Federation of Eugenic Organizations), 1931. Pp. 100. 5½ × 8¼ inches.

LOCKEMANN, GEORG. Bedeutende Männer aus kinderreichen Familien. *Ethik*, Jahrg. 8, pp. 134-151, 1931.

McDOWALL, S. A. Biology and Mankind. *New York* (The Macmillan Co.), 1931. Pp. xviii + 229. 5¼ × 7½ inches. $2.50.

OGBURN, WILLIAM F. The future of man in the light of his past: The viewpoint of a sociologist. *Scientific Monthly*, Vol. XXXII, pp. 294-300, 1931.

II. GROUP BIOLOGY

1. *Environment of Man*

BAKER, O. A., Compiled by. A Graphic Summary of American Agriculture Based Largely on the Census. United States Department of Agriculture Miscellaneous Publication No. 105. *Washington* (U. S. Government Printing Office), 1931. Pp. 228. 5⅞ × 9⅛ inches. 60 cents (paper).

BRANOM, FREDERICK K., Edited by. New Census Atlas of the World. Complete 1930 Census. *Chicago* (The Reilly and Lee Co.), 1931. Pp. iv + 256. 10¼ × 14½ inches. $3.50.

HOWARD, L. O. The Insect Menace. *New York* (The Century Co.), 1931. Pp. xv + 347. 5½ × 8¼ inches. $3.50.

MINISTRY OF COMMERCE AND COMMUNICATIONS. Siam: Nature and Industry 1930. *Bangkok* (Ministry of Commerce and Communications), 1931. Pp. v + 324. 6½ × 9½ inches. Baht 3.00 + 85 satang postage.

2. *Public Health and Hygiene*

BURRITT, BAILEY B. Social service and relief in tuberculous families. *Milbank Memorial Fund Quarterly Bulletin*, Vol. IX, pp. 147-164, 1931.

CHAPIN, CHARLES V. Forty-eighth annual report of the Superintendent of Health of the City of Providence for the year 1930. *Providence* (City Health Department), 1931. Pp. 45 + 3 folding tables. 6 × 9 inches.

DEPARTMENT OF HEALTH, CITY OF NEW YORK, COMMITTEE ON NEIGHBORHOOD HEALTH DEVELOPMENT. Health Center Districts, New York City. Statistical reference data. District populations, school children; local reports of births, infant, tuberculosis and general mortality; tuberculosis and infectious disease registration; public health nursing requirements. Second edition. *New York* (Department of Health), 1931. Pp. xiv + 58 + map. 8½ × 11 inches.

KNOPF, S. ADOLPHUS. Climate in tuberculosis and the prevention of relapses. *Journal of the American Medical Association*, Vol. 96, pp. 2023-2027, 1931.

KNOPF, S. ADOLPHUS. Unemployment and tuberculosis. A suggested program for preventive medical and legal measures. *Medical Journal and Record*, Jan. 7, 1931. Pp. (of reprint) 29.

NEWMAN, SIR GEORGE. On the State of the Public Health. Annual Report of the Chief Medical Officer of the Ministry of Health for the Year 1930. *London* (His Majesty's Stationery Office), 1931. Pp. 261. 6⅛ × 9⅝ inches. 3 shillings net (paper).

OSTENFELD, J., N. HEITMANN and G. NEANDER. Tuberculosis in Denmark, Norway and Sweden. League of Nations, Health Organisation. *Series of League of Nations Publications III. Health. 1931. III. 1. 40 Mt. Vernon St., Boston* (World Peace Foundation); *Geneva* (League of Nations), 1931. Pp. 170. 7¼ × 9½ inches. $1.50 (United States); 6 shillings (Geneva); (paper).

RANDALL, MARIAN G. Public health nursing service in rural families. *Milbank Memorial Fund Quarterly Bulletin*, Vol. IX, pp. 189-203, 1931.

UNITED STATES DEPARTMENT OF LABOR, CHILDREN'S BUREAU. Child Welfare in Selected Counties of Washington. *Bureau Publication No. 206. Washington* (U. S. Government Printing Office), 1931. Pp. v + 111. 6 × 9 inches. 20 cents (paper).

ZIMAND, SAVEL. Campaign calendar of a public health organization. *Milbank Memorial Fund Quarterly Bulletin*, Vol. IX, pp. 165-180, 1931.

3. *Migration*

EUGENICS SURVEY OF VERMONT. Selective migration from three rural Vermont towns and its significance. *Fifth annual report of the Eugenics Survey of Vermont* (Burlington, Vt.), September 1931. Pp. vii + 82 + 3 pages maps. 6 × 9 inches.

PIEKALKIEWICZ, J. L'unification internationale de la statistique des transports sur les voies de navigation intérieure et maritime. *XX^e Session de l'Institut International de Statistique, Madrid,* 1931. II, Rapp., pp. (of reprint) 1-96.

UNION OF SOUTH AFRICA. Statistics of migration 1930. *Pretoria* (Government Printer), 1931. Pp. xv + 52. 9⅝ × 12⅛ inches. 3s. 6d. net (paper).

WILLCOX, W. F. The statistical basis of American "national origins" law restricting immigration. *XX^e Session de l'Institut International de Statistique, Madrid,* 1931. I, Comm., pp. (of reprint) 1-28.

4. *Population*

BURCH, GUY IRVING. Population pressure and war. *Birth Control Review,* Vol. XV, pp. 178-179, 1931.

BURCH, GUY IRVING. The race called American. *Survey Graphic,* April 1931. Pp. (of reprint) 2.

CROCKER, W. R. The Japanese Population Problem. The Coming Crisis. *London* (George Allen and Unwin, Ltd.), 1931. Pp. 240. 5½ × 8½ inches. 10s. 6d. net. [Bibliography of 303 titles.]

EDGE, P. GRANVILLE. Documentos demográficos de España, anteriores al censo. *Boletín Técnico de la Dirección General de Sanidad,* año VI, núm. 6, pp. 1-11, 1931.

GIUSTI, U. Une nouvelle série de monographies de familles agricoles en Italie. *XX^e Session de l'Institut International de Statistique, Madrid,* 1931. III, Comm., pp. (of reprint) 1-10.

KUCZYNSKI, ROBERT R. The Balance of Births and Deaths. Volume II. Eastern and Southern Europe. *Washington* (The Brookings Institution), 1931. Pp. xii + 170. 5¼ × 8 inches. $2.00.

LOTKA, ALFRED J. Orphanhood in relation to demographic factors. A study in population analysis. *Metron,* Vol. IX, pp. 37-109, 1931.

ROESLE, E. Die Lehre vom Lebenstransporteur. *Archiv für Soziale Hygiene und Demographie,* Band V, pp. 113-119, 1930.

SCHEIDT, WALTER. Die rassischen Verhältnisse in Nordeuropa nach dem gegenwärtigen Stand der Forschung. *Stuttgart* (E. Schweizerbart'sche Verlagsbuchhandlung (Erwin Nägele) G. m. b. H), 1930. Pp. 197 + 12 plates + 31 maps. 6½ × 10 inches. 43 marks (paper); 45 marks (cloth). [Bibliography of 354 titles.]

5. *Mortality and General Vital Statistics*

CHAPIN, CHARLES V. Seventy-sixth annual report upon the births, marriages and deaths in the City of Providence for the year 1930, including tables for seventy-five years. *Providence* (City Health Department), 1931. Pp. 127. 6 × 9 inches.

DUBLIN, LOUIS I. New mortality data. Appendicitis, diabetes, pernicious anemia. Statistical charts displayed at the scientific exhibit at the eighty-second session of the American Medical Association, Philadelphia, Pennsylvania, June 8 to 12, 1931. *New York* (Metropolitan Life Insurance Co.), 1931. 13 pp. 6 × 9¼ inches.

EDGE, P. GRANVILLE. The scope and uses of vital records in the tropics. *Proceedings of the Royal Society of Medicine*, Vol. XXIV, pp. 35-43, 1931.

HARPER, ROLAND M. Some Savannah vital statistics of a century ago. *Georgia Historical Quarterly*, Vol. XV, pp. 252-271, 1931.

KURTZ, EDWIN B. Life Expectancy of Physical Property. Based on Mortality Laws. *New York* (The Ronald Press Co.), 1931. Pp. xiv + 205. 5¾ × 8½ inches. $6.00.

LOWRIE, ROBERT J. The maternal mortality rate computed and standardized by a new method. *New York State Journal of Medicine*, October 1, 1931. Pp. (of reprint) 1-24. [Bibliography of 12 titles.]

PRINZING, FRIEDRICH. Handbuch der medizinischen Statistik. Zweite, vollständig umgearbeitete Auflage. *Jena* (Gustav Fischer), 1931. Pp. x + 672. 6½ × 9¼ inches.

REGISTRAR-GENERAL'S Statistical Review of England and Wales for the Year 1930. (New Annual Series, No. 10). Tables. Part I. Medical. *London* (His Majesty's Stationery Office), 1931. Pp. iii + 532. 6 × 9½ inches. 7s. 6d. net (paper).

REGISTRAR-GENERAL'S Statistical Review of England and Wales for the Year 1930. (New Annual Series, No. 10). Tables. Part II. Civil. *London* (His Majesty's Stationery Office), 1931. Pp. iv + 127. 6 × 9⅝ inches. 2 shillings net (paper).

ROESLE, E. Die Abnahme der Krebssterblichkeit im mittleren Alter. *Zeitschrift für Schulgesundheitspflege und soziale Hygiene*, Jahrg. 44, pp. 508-516, 1931.

RUSSELL, W. T. Mortality from cancer according to site in the counties of Scotland, 1923-8. *Journal of Hygiene*, Vol. XXXI, pp. 563-569, 1931.

UNION OF SOUTH AFRICA. Report on the vital statistics of the Union of South Africa 1929. *Pretoria* (Government Printer), 1931. Pp. xxxv + 57. 9⅝ × 12⅛ inches. 3s. 6d. net (paper).

UNITED STATES DEPARTMENT OF COMMERCE, BUREAU OF THE CENSUS. Manual of the International List of Causes of Death. Based on the Fourth Decennial Revision by the International Commission, Paris, October 16 to 19, 1929. *Washington* (U. S. Government Printing Office), 1931. Pp. 342. 5¾ × 9 inches. $1.25.

WIEHL, DOROTHY G. Some recent changes in the mortality among adults. *Journal of Preventive Medicine*, Vol. 4, pp. 215-237, 1930.

6. *Morbidity and Epidemiology*

COLLINS, SELWYN D. The Health of the School Child. A Study of Sickness, Physical Defects and Mortality. With an introduction by Taliaferro Clark. *Public Health Bulletin* No. 200. *Washington* (U. S. Government Printing Office), 1931. Pp. x + 159. 5¾ × 9⅛ inches. 35 cents (paper). [Bibliography of 50 titles.]

FORBER, JANET E. Incurable cancer: An investigation of hospital patients in Eastern London. *Reports on Public Health and Medical Subjects* No. 66. Ministry of Health, London. *London* (His Majesty's Stationery Office), 1931. Pp. 59. 6 × 9⅝ inches. 1s. net (paper).

GREENWOOD, MAJOR. President's Address, Section of Epidemiology and State Medicine. *Proceedings of the Royal Society of Medicine*, Oct. 23, 1931. pp. 1-6.

GREENWOOD, M., W. W. C. TOPLEY, and J. WILSON. Contributions to the experimental study of epidemiology. Further observations on the effect of vaccination on herd mortality. *Journal of Hygiene*, Vol. XXXI, pp. 484-492, 1931. [Bibliography of 3 titles.]

KORNS, JOHN H. Tuberculous infection among school children in a rural area in New York State. *American Review of Tuberculosis*, Vol. XXIV, pp. 532-536, 1931.

LEAGUE OF NATIONS, HEALTH ORGANISATION. Statistiques des Maladies à Déclaration Obligatoire pour l'Année 1929, Publiées par la Section d'Hygiène. (Statistics of Notifiable Diseases for the Year 1929, Published by the Health Section). *40 Mt. Vernon St., Boston* (World Peace Foundation), 1931. Pp. 81. 9⅝ × 12½ inches. 80 cents (paper). [In French and English.]

ROESLE, E. Gesundheitsbarometer. *Allgemeines Statistisches Archiv*, Band 21, pp. 328-342, 1931.

SORSBY, MAURICE. Cancer and Race. A Study of the Incidence of Cancer Among Jews. Conducted under the auspices of the Jewish Health Organisation of Great Britain. With a Preface by F. E. Fremantle. *New York* (William Wood and Co.), 1931. Pp. xvi + 120. 5¼ × 8¼ inches. $3.00 net.

7. *Natality, Fecundity, Fertility*

BOLDRINI, MARCELLO. Fertilità e intelligenza. *Rivista Internazionale di Scienze Sociali e Discipline Ausiliarie*, Anno 39, Vol. II, Fasc. I, pp. 3-21, 1931.

CASTALDI, LUIGI. Relazione sull'indagine di mille genitori di famiglie numerose nella città di Cagliari. *Atti della Società fra i Cultori delle Scienze Mediche e Naturali in Cagliari*, Fasc. V, pp. (of reprint) 1-19, 1931. [Bibliography of 18 titles.]

HANKINS, FRANK H. Civilization and fertility. Has the reproductive power of western peoples declined? *Eugenics Review*, Vol. XXIII, pp. 145-150, 1931.

MOENCH, G. L., and HELEN HOLT. Sperm morphology in relation to fertility. *American Journal of Obstetrics and Gynecology*, Vol. XXII, pp. 199-210, 1931. [Bibliography of 37 titles.]

NOTESTEIN, FRANK W. The decrease in size of families from 1890 to 1910. *Milbank Memorial Fund Quarterly Bulletin*, Vol. IX, pp. 181-188, 1931.

ROESLE, E. Geburtshilfliche Statistik im Staate Pennsylvania. *Archiv für Soziale Hygiene und Demographie*, Band IV, pp. 128-129, 1929.

ROESLE, E. Der jahreszeitliche Verlauf von Geburten und Konzeptionen. *Archiv für Soziale Hygiene und Demographie*, Band V, pp. 509-510, 1930.

SANDERS, J. The Declining Birth Rate in Rotterdam. A Statistical Analysis of the Drop in the Number of Children in 24,664 Rotterdam Families During the last 50 Years. *The Hague* (Martinus Nijhoff), 1931. Pp. x + 179. 6½ × 10 inches. 6 guilders (paper).

8. *Birth Control*

BURCH, GUY I. The pertinent question. *Birth Control Review*, Vol. XV, pp. 258-260, 1931.

DE GUCHTENEERE, R. Judgment on Birth Control. *New York* (The Macmillan Co.), 1931. Pp. 224. 5⅛ × 7⅜ inches. $2.00.

HIMES, NORMAN E. Note on the early history of contraception in America. *New England Journal of Medicine*, Vol. 205, pp. 438-440, 1931.

HIMES, NORMAN E. Soranus on birth control. (Second century A. D.). *New England Journal of Medicine*, Vol. 205, pp. 490-491, 1931.

HIMES, NORMAN E. The Truth About Birth Control with a Bibliography of Birth-Control Literature. *New York* (The John Day Co.), 1931. Pp. 28. 5¼ × 7¾ inches. 25 cents (35 cents in Canada) (paper). [Bibliography of 10 titles.]

HOLLAND-RANTOS COMPANY. A selected bibliography on the technique of contraception and social aspects of birth control. *New York* (Holland-Rantos Co., Research Dept.), 1931. Pp. 20. 3⅜ × 6¼ inches.

KNOPF, S. ADOLPHUS. The dilemma of the family physician regarding contraception and sterilization for race betterment. *The Medical Times*, April 1930, pp. 3-14.

KNOPF, S. ADOLPHUS. The present status of birth control in England and the U. S. Recent developments. *Medical Journal and Record*, Vol. CXXXIV, pp. 105-109, 171-174, 224-226, 1931. [Bibliography of 12 titles.]

MOORE, EDWARD R. The Case Against Birth Control. *New York* (The Century Co.), 1931. Pp. x + 311. 5 × 7½ inches. $2.50. [Bibliography of 75 titles.]

SANGER, MARGARET. My Fight for Birth Control. *New York* (Farrar and Rinehart), 1931. Pp. vii + 360. 5¾ × 8⅝ inches. $3.00.

9. *Marriage and Divorce*

DICKINSON, ROBERT L., and LURA BEAM. A Thousand Marriages. A Medical Study of Sex Adjustment. (With a Foreword by Havelock Ellis.) *Baltimore* (The Williams and Wilkins Co.), 1931. Pp. xxv + 482. 6 × 9 inches. $5.00.

SPAETH, LOUISE M. Marriage and Family Life Among Strange Peoples. Studies of Actual Savage Life. *Chicago* (Thomas S. Rockwell Co.), 1931. Pp. 111. 5½ × 8 inches. $1.25 net.

III. BEHAVIOR

1. *Racial Psychology*

CRAWLEY, ERNEST. Dress, Drinks, and Drums. Further Studies of Savages and Sex. Edited by Theodore Besterman. *London* (Methuen and Co., Ltd.), 1931. Pp. x + 274. 5½ × 8¾ inches. 12s. 6d. net.

KING, VERONICA and PAUL. Looking Inwards. *London* (Heath Cranton, Ltd.), 1931. Pp. 209. 5½ × 8¾ inches. 7s. 6d. net, postage 6d. extra.

LILLIE, RALPH S. Science and life. *Journal of Philosophy*, Vol. XXVII, pp. 421-430, 1930.

PARMELEE, MAURICE. Nudism in Modern Life. The New Gymnosophy. With an Introduction by Havelock Ellis. *New York* (Alfred A. Knopf, Inc.), 1931. Pp. xiv + 303. 5½ × 8⅛ inches. $3.00. [31 pages of Notes and References.]

RUSSELL, BERTRAND. The Scientific Outlook. *New York* (W. W. Norton and Co., Inc.), 1931. Pp. 277. 5¾ × 8¼ inches. $3.00.

STEGGERDA, MORRIS. Results of psychological tests given to Maya Indians in Yucatan. *Eugenical News*, Vol. XVI, pp. 120-125, 1931. [Bibliography of 9 titles.]

2. *Individual Psychology*

ACKERSON, LUTON. Children's Behavior Problems. A Statistical Study Based upon 5,000 Children Examined Consecutively at the Illinois Institute for Juvenile Research. I. Incidence, Genetic and Intellectual Factors. *Chicago* (University of Chicago Press), 1931. Pp. xxi + 268. 6 × 9 inches. $4.00.

ADAMS, GRACE. Psychology: Science or Superstition? *New York* (Covici, Friede, Inc.), 1931. Pp. 299. 5 × 8¼ inches. $2.50. [Bibliography of 80 titles.]

DORSEY, GEORGE A. Man's Own Show: Civilization. *New York* (Harper and Brothers), 1931. Pp. xvi + 977. 6¼ × 9⅜ inches. $5.00.

FOX, CHARLES. The Mind and Its Body. The Foundations of Psychology. *New York* (Harcourt, Brace and Co.), 1932. Pp. xii + 316. 5½ × 8½ inches. $3.50. [Bibliography of 169 titles.]

HALVERSON, H. M. An Experimental Study of Prehension in Infants by Means of Systematic Cinema Records. *Genetic Psychology Monographs*, Volume X, Nos. 2-3. *Worcester, Mass.* (Clark University Press), 1931. Pp. 180. 6½ × 9¾ inches. $3.00 (paper).

HEINRICH, W. Les fonctions des capillaires en rapport avec la fixation de l'attention. Note préliminaire. *Bulletin de l'Académie Polonaise des Sciences et des Lettres, Classe des Sciences Mathématiques et Naturelles—Série B: Sciences Naturelles (II)*, pp. 55-58, 1929.

HEINRICH, W., and T. STRZEMBOSZ. Les fonctions des capillaires et la concentration de l'attention. *Bulletin de l'Académie Polonaise des Sciences et des Lettres, Classe des Sciences Mathématiques et Naturelles. Série B: Sciences Naturelles (II)*, pp. 321-349, 1931.

LILLIE, RALPH S. Types of physical determination and the activities of living organisms. *Journal of Philosophy*, Vol. XXVIII, pp. 561-573, 1931.

NISSEN, HENRY W. A Field Study of the Chimpanzee. Observations of Chimpanzee Behavior and Environment in Western French Guinea. *Comparative Psychology Monographs*, Vol. 8, No. 1, Serial No. 36. *Baltimore* (The Johns Hopkins Press), 1931. Pp. vi + 122. 6⅞ × 10 inches. $1.75. [Bibliography of 9 titles.]

SKAGGS, ERNEST B. The Major Forms of Inhibition in Man. *Chicago* (The University of Chicago Press), 1931. Pp. vii + 104. 6 × 9 inches. $1.50 (paper).

STRONG, EDWARD K., JR. Change of Interests with Age. Based on Examination of More Than Two Thousand Men Between the Ages of Twenty and Sixty Representing Eight Occupations. *Stanford University, Calif.* (Stanford University Press), 1931. Pp. xix + 235. 5⅞ × 8¼ inches. $4.00.

SWIFT, EDGAR J. The Jungle of the Mind. *New York* (Charles Scribner's Sons), 1931. Pp. x + 340. 5½ × 8 inches. $2.50.

3. *Psychiatry*

ALLAIX, H. De l'Inversion sexuelle à la Formation et à la Détermination des Sexes. *Paris* (J. Peyronnet et Cie), 1931. Pp. vii + 85. 6½ × 9¾ inches.

[ANON.] Annual statistical review of patients with mental disease in the state hospitals and licensed institutions, 1930. *Forty-second Annual Report of the Department of Mental Hygiene, New York State* (Albany, N. Y.), pp. 158-292, 1931.

BERRY, RICHARD J. A., and R. G. GORDON. The Mental Defective. A Problem in Social Inefficiency. *New York* (Whittlesey House, McGraw-Hill Book Co., Inc.), 1931. Pp. xiii + 225. 5½ × 8 inches. $2.50.

FISCHER, MAX. Psychiater und organisierte Alkoholbekämpfung. *Psychiatrisch-Neurologische Wochenschrift*, Nr. 13. Pp. (of reprint) 4, 1931.

4. *History and Biography*

CAMPBELL, ANNA M. The Black Death and Men of Learning. *New York* (Columbia University Press), 1931. Pp. xii + 210. 5½ × 8 inches. $3.00. [Bibliography of 245 titles.]

DAVIS, WATSON, Edited by. Science Today. A Layman's Handbook of Recent Discoveries by Various Eminent Men of Science. *New York* (Harcourt, Brace and Co.), 1931. Pp. ix + 310. 5¼ × 8 inches.

EMBREE, EDWIN R. Brown America. The Story of a New Race. *New York* (The Viking Press), 1931. Pp. vi + 311. 5½ × 8⅛ inches. $2.50. [Books by and about the Negro: 70 titles.]

GIPSON, LAWRENCE H. Studies in Colonial Connecticut taxation. 1. The taxation of the Connecticut towns, 1750-1775. 2. Connecticut taxation and parliamentary aid preceding the Revolutionary War. *Lehigh University Publication* Vol. V, No. 11, *The Institute of Research* Circular No. 65, Studies in the Humanities No. 11. *Bethlehem, Pa.* (Lehigh University), 1931. Pp. 35. 5¾ × 8½ inches. 20 cents (paper).

GREGORY, JOSHUA C. A Short History of Atomism from Democritus to Bohr. *London* (A. and C. Black, Ltd.); *New York* (The Macmillan Co.), 1931. Pp. 258. 5½ × 8½ inches. 10s. 6d. net (England); $3.50 (U. S. A.).

KNOPF, S. ADOLPHUS. In Memoriam. James Freyer Cooper. *Medical Journal and Record*, May 20, 1931. Pp. (of reprint) 1-7.

POWER, SIR D'ARCY. The Foundations of Medical History. *Baltimore* (The Williams and Wilkins Co.), 1931. Pp. x + 182. 4¾ × 7¼ inches. $3.00.

ROBINSON, VICTOR. Pathfinders in Medicine. *New York* (Medical Life Press), 1929. Pp. xvii + 810. 6¼ × 9¼ inches. $10.00.

SARTON, GEORGE. Introduction to the History of Science. Volume II. From Rabbi Ben Ezra to Roger Bacon. (In two parts). Carnegie Institution of Washington Publication No. 376. *Baltimore* (The Williams and Wilkins Co.), 1931. Pp. xxxv + xvi + 1251. 7 × 10 inches. $12.00.

SINGER, CHARLES. The Story of Living Things. A Short Account of the Evolution of the Biological Sciences. *New York* (Harper and Brothers), 1931. Pp. xxxv + 572. 5¾ × 8⅜ inches. $5.00.

SINGER, CHARLES. Sketches in the history of English medicine. I. Dawn: Celtic and Anglo-Saxon medical and pharmaceutical practice from the 9th to the 13th centuries. *Chemist and Druggist*, June 30, 1928, pp. 825-832. [Reprinted in Annual Special Issue, Vol. CXII, No. 2629, June 28, 1930.]

SINGER, CHARLES. Sketches in the history of English medicine. II. Twilight: The Anglo-Norman period and the advent of learning of Salerno and the Arabs (13th and 14th centuries). *Chemist and Druggist*, June 29, 1929, pp. 795-804. [Reprinted in Annual Special Issue, Vol. CXII, No. 2629, June 28, 1930.]

SINGER, CHARLES. Sketches in the history of English medicine. III. Daybreak: The later Middle Ages and the rise of new learning. *Chemist and Druggist*, December 21, 1929, pp. 741-748. [Reprinted in Annual Special Issue, Vol. CXII, No. 2629, June 28, 1930.]

SINGER, CHARLES. Sketches in the history of English medicine. IV. Morning Light: The seventeenth century and the beginnings of the scientific method—Harvey, Paré, Sydenham and the Royal Society. *Chemist and Druggist*, June 28, 1930, pp. 799-808. [Reprinted in Annual Special Issue, Vol. CXII, No. 2629, June 28, 1930.]

SINGER, CHARLES. Sketches in the history of English medicine. V. Daylight: The eighteenth and nineteenth centuries—the medical schools, surgery and public health—Boerhaave, the Hunters, Pringle, Lind, Jenner, Chadwick, and Simon. *Chemist and Druggist*, Annual Special Issue, Vol. CXII, No. 2629, pp. 783-790, June 28, 1930.

SWEET, HENRY. The History of Language. The Temple Cyclopædic Primers. *London and Toronto* (J. M. Dent and Sons, Ltd.), undated. Pp. xi + 148. 3¾ × 6 inches. 1s. 9d. net. [Bibliography of 48 titles.]

THAYER, WILLIAM S. Osler and Other Papers. *Baltimore* (The Johns Hopkins Press), 1931. Pp. 386. 5¾ × 8¾ inches. $3.50.

THE ROCKEFELLER FOUNDATION. Annual Report 1930. *New York* (The Rockefeller Foundation), 1931. Pp. viii + 380. 5½ × 8½ inches.

WIEGERS, F., Editor, with Collaboration of F. Bork, H. Plischke, Bruno K. Schultz, and L. Wolff. Herman Wirth und die deutsche Wissenschaft. *München* (J. F. Lehmanns Verlag), 1932. Pp. 69. 6 × 9 inches. 2.50 marks (paper).

WRIGHT, HORACE W. The age of Roman sacrificial victims. *Lehigh University Publication*, Vol. V, No. 11. *The Institute of Research Circular* No. 66. *Studies in the Humanities*, No. 12. *Bethlehem, Pa.* (Lehigh University), 1931. Pp. 23. 6 × 9 inches. 10 cents (paper).

5. *Sociology, Law, Politics, and Religion*

ACUNA, CHILE. Women for Sale. *New York* (William Godwin, Inc.), 1931. Pp. xii + 201. 5 × 7½ inches. $2.00.

ASSOCIATED CHAMBERS OF COMMERCE OF CHINA. A message to the people of the world. Pp. 4. No date or place of publication.

CARNEGIE ENDOWMENT FOR INTERNATIONAL PEACE. Text of the draft convention for the Disarmament Conference. *International Conciliation*, No. 275. *Worcester, Mass.* (Carnegie Endowment for International Peace), 1931. Pp. 55. 5 × 7¾ inches. 5 cents (paper).

CHURCH, SAMUEL H. The Liberal Party in America. Its Principles and its Platform. *New York* (G. P. Putnam's Sons), 1931. Pp. vi + 124. 5 × 7⅛ inches. $1.50.

COTTON, EDWARD H., Gathered and Edited by. Has Science Discovered God? A Symposium of Modern Scientific Opinions. *New York* (Thomas Y. Crowell Co.), 1931. Pp. lviii + 308. 5¾ × 8⅝ inches. $3.50.

EOM. Rededication of the principles of the Declaration of Independence for the fulfilment of the destiny of the United States. "Liberty for all humanity." *Chicago* (EOM, Suite 1008-9 Auditorium Bldg., 431 So. Wabash Ave.), 1931. Pp. 9. 5¾ × 9 inches.

FISCHER, MAX. Ärzteschaft und organisierte Arbeit gegen den Alkoholismus. *Deutsches Ärzteblatt*, Nr. 12 vom 21, April, 1931. Pp. (of reprint) 2.

FROST, HENRY W. Miraculous Healing. A Personal Testimony and Biblical Study. *New York* (Richard R. Smith, Inc.), 1931. Pp. vii + 175. 5 × 7⅜ inches. $1.50.

GARRETT, GARET. "The rescue of Germany" and "As noble lenders." Reprinted by special permission from *The Saturday Evening Post* as "O. P. M. Other people's money! The greatest American racket." *New York* (The Chemical Foundation, Inc.), 1931. Pp. 103. 5 × 7½ inches.

HAUPTMANN, HANS. Erneuerung aus Blut und Boden: Die Lappobewegung der finnischen Bauernschaft, ein Weg zur Befreiung vom Bolschewismus. *München* (J. F. Lehmanns Verlag), 1932. Pp. 76. 6 × 9 inches. 2.50 marks (paper).

HUBBARD, URSULA P. The coöperation of the United States with the League of Nations and with the International Labour Organization. *International Conciliation*, No. 274. *Worcester, Mass.* (Carnegie Endowment for International Peace), 1931. Pp. 159. 5 × 7¾ inches. 5 cents (paper).

JAFFIN, GEORGE H. Theorems in Anglo-American labor laws. *Columbia Law Review*, Vol. XXXI, pp. 1104-1134, 1931.

JESSUP, PHILIP C. The United States and the Permanent Court of International Justice. The acceptance of the Senate reservations. The documentary record of the negotiations for the accession of the United States to the Court. *International Conciliation* (Carnegie Endowment for International Peace, Worcester, Mass.), No. 273, pp. 589-670, 1931. 5 cents (paper).

PEARL, RAYMOND. Some notes on the census of religious bodies, 1926. *Journal of Social Psychology*, Vol. 2, pp. 417-432, 1931. [Bibliography of 7 titles.]

PROCEEDINGS of public mass meetings in New York City and Washington, D. C. Armistice Day, November 11, 1931, to prepare for the Disarmament Conference to be held in Geneva, February, 1932. *International Conciliation Special Bulletin. Worcester, Mass.* (Carnegie Endowment for International Peace), 1931. Pp. 59. 5 × 7¾ inches. 5 cents (paper).

REED, JAMES A. The Rape of Temperance. *New York* (Cosmopolitan Book Corporation), 1931. Pp. vii + 307. 5 × 7⅜ inches. $2.00.

UNITED STATES DEPARTMENT OF JUSTICE. Bureau of Investigation. Published for the information of law-enforcement officials and agencies. *Washington* (U. S. Government Printing Office), 1931. Pp. 8. 5⅞ × 9 inches.

UNITED STATES DEPARTMENT OF LABOR, CHILDREN'S BUREAU. Juvenile-Court Statistics, 1929, Based on Information Supplied by 96 Courts. Third Annual Report. *Bureau Publication* No. 207. *Washington* (U. S. Government Printing Office), 1931. Pp. ii + 61. 6 × 9 inches. 15 cents (paper).

6. *Economics*

CRAIG, J. I. Statistics of consumption and of annual stocks-in-hand. *XX^e Session de l'Institut International de Statistique, Madrid,* 1931. II, Comm., pp. (of reprint) 1-7. [Résumé in French.]

DUBLIN, LOUIS I., and W. A. BERRIDGE. The need for a new survey of family budgets and buying habits. Two points of view: I. Family budgets from a general welfare point of view, by Louis I. Dublin; II. Family budgets from an economic and merchandising point of view, by William A. Berridge. Adapted from articles appearing in *The New York Sunday Times,* May 10, 1931, and *The Annalist,* July 17, 1931. *Metropolitan Life Insurance Co., Reprint.* Pp. (of reprint) 1-22.

HILTON, J. Tables for report on real wages and unemployment. *XX^e Session de l'Institut International de Statistique, Madrid,* 1931. III. Rapp., pp. (of reprint) 1-35.

MORTARA, GIORGIO. Sommario di Statistica. *Milan* (Università Bocconi di Milano), 1931-IX. Pp. xv + 393. 7 × 10 inches. 40 lire (paper).

NEW YORK, STATE OF. Annual Report of the State Tax Commission, 1930. *Legislative Document* (1931), No. 11. *Albany, N. Y.* (State Tax Commission), 1931. Pp. 418. 5⅝ × 8¾ inches.

PIDGEON, MARY E. Women in Industry. A Series of Papers to Aid Study Groups. *Bulletin of the Women's Bureau* No. 91. *Washington* (U. S. Government Printing Office), 1931. Pp. viii + 79. 5⅜ × 9⅛ inches. 15 cents (paper).

PIEKALKIEWICZ, J. Le service statistique des banques de crédit agricole hypothécaire. *XX^e Session de l'Institut International de Statistique, Madrid,* 1931. II, Comm., pp. (of reprint) 1-81.

PLATZER, H. Zur Statistik der Fertigwarenpreise. *XX^e Session de l'Institut International de Statistique, Madrid,* 1931. II, Rapp., pp. (of reprint) 1-10. [Résumé in French.]

REDMOND, OLNEY. The Olney Redmond Plan to End Unemployment and Depression Permanently. A Job for Every Man! *Schenectady, N. Y.* (Olney Redmond), 1931. Pp. xi + 287. 5¼ × 7½ inches. $2.00.

7. *Education*

GRUENBERG, BENJAMIN C. Complacency and specialization. A challenge to teachers of science. *Progressive Education,* October, 1931, pp. (of reprint) 5.

GRUENBERG, BENJAMIN C. Educational exploitation of errors. *Educational Method,* October 1931, pp. 9-16.

HULL, CALLIE, and CLARENCE J. WEST. Doctorates Conferred in the Sciences by American Universities, 1930-1931. Compiled for the Research Information Service, National Research Council. *Reprint and Circular Series of the National Research Council* No. 101. *Washington* (National Research Council), 1931. Pp. 55. 6⅝ × 9⅜ inches. 50 cents (paper).

OWRE, ALFRED. Dental education as related to medical education. *Journal of the Association of American Medical Colleges* (in print). Pp. (of reprint) 13. 1931.

OWRE, ALFRED. Discussion of paper on "Dental Education," by Frank M. Casto. [No place of publication], pp. 385-403, 1931.

OWRE, ALFRED. Possible educational solutions of the social-economic problems of the professions of medicine and dentistry. *Journal of Dental Research*, Vol. XI, No. 5, October, 1931. Pp. (of reprint) 17.

OWRE, ALFRED. To the prospective dental student. An enlargement of a radio talk in a series on choosing a vocation, May 21, 1930. Published by the *University of the State of New York, State Education Department*, "Dentistry as a Vocation." Pp. (of reprint) 9, 1931.

8. *Ethnology, Cultural Anthropology, Travel and Exploration*

BARRETT, ROBERT and KATHERINE. A Yankee in Patagonia: Edward Chace. With Frontispiece and Introduction by Rockwell Kent. *New York* (Houghton Mifflin Co.), 1931. Pp. ix + 349. 5¾ × 8½ inches. $4.00.

BRYK, FELIX. Die Beschneidung bei Mann und Weib. Ihre Geschichte, Psychologie und Ethnologie. *Neubrandenburg* (Gustav Feller), 1931. Pp. x + 319. 6¼ × 9¼ inches. 15.60 marks (paper); 18 marks (bound). [Bibliography of 287 titles.]

DEUEL, JOHN V. Indians, Crocodiles and Monkeys. *New York* (The Century Co.), 1931. Pp. viii + 247. 5 × 7½ inches. $2.00.

GREGORY, J. W. Race as a Political Factor. Conway Memorial Lecture, Delivered at Conway Hall, Red Lion Square, W. C. 1, on April 15, 1931. *London* (Watts and Co.), 1931. Pp. 72. 4 × 6½ inches. 2 shillings net.

GUENTHER, KONRAD. A Naturalist in Brazil. The Record of a Year's Observation of her Flora, her Fauna, and her People. Translated by Bernard Miall. *Boston and New York* (Houghton Mifflin Co.), 1931. Pp. 400. 6¼ × 9½ inches. $5.00.

HARGRAVE, LYNDON L. The influence of economic geography upon the rise and fall of the Pueblo culture in Arizona. *Museum Notes* (*Museum of Northern Arizona, Flagstaff*), Vol. 4, No. 6, pp. 1-3, 1931. 10 cents (paper).

HENDERSON, G. C. Fiji and the Fijians, 1835-1856. *Sydney, Australia* (Angus and Robertson, Ltd.), 1931. Pp. xv + 333. 6 × 9¼ inches. $5.00. [One chapter of bibliography.]

LESLIE, LIONEL A. D. Wilderness Trails in Three Continents. An Account of Travel, Big Game Hunting and Exploration in India, Burma, China, East Africa and Labrador. *London* (Heath Cranton, Ltd.), 1931. Pp. xvi + 223. 5⅜ × 8½ inches. 10s. 6d. net (postage 6d. extra).

MCSPADDEN, J. WALKER. To the Ends of the World and Back. Scouting for a Great Museum. *New York* (Thomas Y. Crowell Co.), 1931. Pp. xv + 362. 5½ × 8⅛ inches. $3.00.

MILLER, JANET. Jungles Preferred. *New York* (Houghton Mifflin Co.), 1931. Pp. 321. 5½ × 8 inches. $3.50.

NORDEN, HERMANN. A Wanderer in Indo-China. The Chronicle of a Journey Through Annam, Tong-King, Laos, and Cambodgia, with Some Account of Their People. *London* (H. F. and G. Witherby), 1931. Pp. 290. 5½ × 8½ inches. 15 shillings net.

SCHWEITZER, ALBERT. On the Edge of the Primeval Forest. Experiences and Observations of a Doctor in Equatorial Africa. Translated by C. T. Campion. *New York* (The Macmillan Co.), 1931. Pp. 180. 5 × 7½ inches. $2.00.

THILENIUS, GEORG. On some biological view-points in ethnology. (The Huxley Memorial Lecture for 1931). *Journal of the Royal Anthropological Institute*, Vol. LXI, pp. 287-299, 1931.

WALDRON, MALCOLM. Snow Man. John Hornby in the Barren Lands. *New York* (Houghton Mifflin Co.), 1931. Pp. x + 292. 5½ × 8 inches. $3.00.

IV. SOMATOLOGY AND CONSTITUTION

1. *Anatomy*

FISCHEL, ALFRED. Grundriss der Entwicklung des Menschen. *Vienna and Berlin* (Julius Springer), 1931. Pp. vi + 141. 6¾ × 10¼ inches. 11 marks (paper); 12.60 marks (cloth).

GALLAUDET, B. B. A Description of the Planes of Fascia of the Human Body. With Special Reference to the Fascia of the Abdomen, Pelvis and Perineum. *New York* (Columbia University Press), 1931. Pp. 75. 5⅜ × 8 inches. $2.00.

GLAISTER, JOHN. A Study of Hairs and Wools Belonging to the Mammalian Group of Animals, Including a Special Study of Human Hair Considered from the Medico-Legal Aspect. Egyptian University, Faculty of Medicine, Publication No. 2. *Cairo* (Misr Press), 1931. Pp. 188 + 145 plates. 7¾ × 11 inches.

KUNTZ, ALBERT. A Text-Book of Neuro-Anatomy. *Philadelphia* (Lea and Febiger), 1931. Pp. 359. 5⅞ × 9¼ inches. $5.50 net. [Bibliography of 345 titles.]

LEWIS, WARREN H. A human tubal egg, unfertilized. *Bulletin of the Johns Hopkins Hospital*, Vol. XLVIII, pp. 368-372, 1931. [Bibliography of 6 titles.]

MCEWEN, ROBERT S. Vertebrate Embryology. Revised. *New York* (Henry Holt and Co.), 1931. Pp. xi + 581. 5½ × 8½ inches. $4.75. [Bibliography at end of each chapter.]

RICHARDS, AUTE. Outline of Comparative Embryology. *New York* (John Wiley and Sons, Inc.), 1931. Pp. 444. 5¾ × 9 inches. $5.00. [Bibliography of 612 titles.]

2. *Physical Anthropology and Anthropometry*

BASLER, ADOLF. Der Schwerpunkt des lebenden Menschen. Schwerpunktwage und Schwerpunktpendel. *Leipzig* (Gustav Fock); *Canton* (Verlag der Sun Yatsen-Universitaet), 1931. Pp. 100. 7 × 10½ inches. 9 marks (paper).

BATTLEY, HARRY. Single Finger Prints. A New and Practical Method of Classifying and Filing Single Finger Prints and Fragmentary Impressions. *New Haven* (Yale University Press), 1931. Pp. 98. 6 × 9½ inches. $2.50.

BEAN, ROBERT B. Stature in old Virginians. *American Journal of Physical Anthropology*, Vol. XV, pp. 355-419, 1931. [Bibliography of 377 titles.]

FISCHER, EUGEN. Sind die alten Kanarier ausgestorben? Eine anthropologische Untersuchung auf den Kanarischen Inseln, ausgeführt mit Hilfe der Notgemeinschaft der Deutschen Wissenschaft. *Zeitschrift für Ethnologie*, Jahrg. 62, pp. 258-281, 1930.

GRAY, HORACE, and J. G. AYRES. Growth in Private School Children, with Averages and Variabilities Based on 3,110 Measurings on Boys, and 1,473 on Girls, from the Ages of One to Nineteen Years. *Chicago* (University of Chicago Press), 1931. Pp. xv + 282. 6 × 9 inches. $3.50. [Bibliography of 23 pages.]

NYÈSSEN, D. J. H. Somatical Investigation of the Javanese 1929. *Bandoeng* (Anthropological Laboratory of Java), 1931 (?). Pp. x + 119 + 42 plates + 3 pp. appendices. 6⅛ × 9½ inches.

3. *Constitution*

BRANDT, WALTER. Grundzüge einer Konstitutions-Anatomie. *Berlin* (Julius Springer), 1931. Pp. ii + 382. 6¾ × 10⅛ inches. 28 marks (paper); 29.80 marks (cloth). [Bibliography of 32 pages.]

JACQUELIN, ANDRÉ. Essai d'interprétation des types morphologiques chez les tuberculeux pulmonaires. *Bulletin de la Société d'Étude des Formes Humaines*, 8e Année, pp. 171-193, 1930.

V. PHYSIOLOGY AND BIOCHEMISTRY

1. *Physiology*

BAINBRIDGE, F. A. The Physiology of Muscular Exercise. Third Edition, Rewritten by A. V. Bock and D. B. Dill. *New York* (Longmans, Green and Co.), 1931. Pp. viii + 272. 5¾ × 8¾ inches. $5.00. [Bibliography of 418 titles.]

BARDIER, E., G. BATTEZ, H. BIERRY, LÉON BINET, P. CARNOT, P. CONBEMALE, C. DELEZENNE, A. DESGREZ, R. GAYET, R. GLÉNARD, L. HALLION, M. LISBONNE, A. PI SUNER, M. VAGLIANO, E. WOLLMAN. Traité de Physiologie Normale et Pathologique. Tome II. Alimentation et Digestion. *Paris* (Masson et Cie), 1931. Pp. 565. 6½ × 9½ inches. 100 francs.

CHESLOCK, LOUIS. Introductory Study on Violin Vibrato. Research Studies in Music, Department of Research of the Conservatory of Music, Number 1. *Baltimore* (Peabody Conservatory of Music), 1931. Pp. 79. 6¾ × 9¾ inches. $1.00 (paper). [Bibliography of 16 titles.]

CREW, F. A. E. Pregnancy Diagnosis Station. Report on first year's working. *British Medical Journal*, April 5, 1930, pp. 662-664.

CREW, F. A. E. Puberty and maturity. Being the Presidential address delivered at the Second International Congress for Sex Research held in London from 3rd to 9th August. *Edinburgh and London* (Oliver and Boyd), 1930. Pp. (of reprint) 1-19. 5½ × 8½ inches. [Bibliography of 22 titles.]

FOUCHER, ROBERT. The Problem of Life. *1219 Eye St., N.W., Washington* (Robert Foucher), 1931. Pp. 24. 6 × 9 inches. $1.00 (paper).

GRAVES, WILLIAM P. Female Sex Hormonology. A Review. *Philadelphia* (W. B. Saunders Co.), 1931. Pp. 131. 5¾ × 8⅞ inches. $3.50. [Bibliography of 116 titles.]

GREENWOOD, A. W., Edited by. Proceedings of the Second International Congress for Sex Research, London, 1930. *London* (Oliver and Boyd), 1931. Pp. xi + 637. 5½ × 8¾ inches. 21 shillings net.

LEPESCHKIN, W. W. The effect of sunlight on human blood cells. *Science*, Vol. 73, p. 568, 1931.

LOEWY, A. Über Klimatophysiologie. *Leipzig* (Georg Thieme), 1931. Pp. 77. 5¾ × 8½ inches. 4.80 marks (paper). [Bibliography of 28 titles.]

NEW YORK COMMISSION ON VENTILATION. School Ventilation. Principles and Practices. Final Contribution of the New York Commission on Ventilation whose work was financed by the Milbank Memorial Fund 1913-1923, 1926-1929. *New York* (Bureau of Publications, Teachers College, Columbia University), 1931. Pp. viii + 73. 5 × 7⅜ inches. $1.00. [Bibliography of 37 titles.]

WEISS, PAUL. Aus den Werkstätten der Lebensforschung. *Berlin* (Julius Springer), 1931. Pp. 192. 4½ × 7¼ inches. 4.80 marks.

WESTON, WILLIAM. Vitamin chart. Prepared as a scientific exhibit, American Medical Association, Minneapolis, Minnesota, June 11-15, 1928. Revised June, 1931, in collaboration with the author by Harold Levine. *Boston* (E. L. Patch Co.), 1931. 4 pp. [Reference list of 15 titles.]

2. *Senescence, Senility and Longevity*

HARPER, ROLAND M. Some new light on the inheritance of longevity. *Journal of Heredity*, Vol. XXII, pp. 93-98, 1931.

LEPESCHKIN, W. W. Death and its causes. *Quarterly Review of Biology*, Vol. VI, pp. 167-177, 1931.

OLIVER, JAMES. Food and the natural limit of man's life. *Medical Press and Circular*, October 14, 1931. Pp. (of reprint) 1-15.

RUBINOW, I. M., Edited by. The Care of the Aged. Proceedings of the Deutsch Foundation Conference 1930. Social Service Monographs, Number Fourteen. *Chicago* (University of Chicago Press), 1931. Pp. xiii + 144. 6 × 9 inches. $2.00.

SCHMIDT, PETER. The Conquest of Old Age. Methods to Effect Rejuvenation and to Increase Functional Activity. Translated by Eden and Cedar Paul. *New York* (E. P. Dutton and Co.), 1931. Pp. xvii + 313 + 40 plates. $5.00.

VECKI, VICTOR G. Prevention of Premature Senility. *Boston* (The Stratford Co.), 1931. Pp. 127. 4⅞ × 7⅜ inches. $1.00.

WILLIUS, FREDERICK A. The heart in old age. A study of 700 patients seventy-five years of age and older. *American Journal of the Medical Sciences*, Vol. CLXXXII, pp. 1-8, 1931. [Bibliography of 6 titles.]

3. *Biochemistry*

GILLESPIE, LOUIS J. Physical Chemistry. An Elementary Text, Primarily for Biological and Pre-medical Students. *New York* (McGraw-Hill Book Co.), 1931. Pp. ix + 287. 5½ × 8 inches.

JOYET-LAVERGNE, PH. La Théorie Physico-Chimique de la Sexualité. *Paris* (Gaston Doin et Cie), 1932. Pp. 103. 4½ × 6⅞ inches. 10 francs (paper).

4. *Pharmacology*

CHEN, K. K. Researches on Chinese materia medica. *Journal of the American Pharmaceutical Association*, Vol. XX, pp. 110-113, 1931.

HAMMETT, FREDERICK S. The proliferative reaction of the skin to sulfhydryl and its biological significance. *Protoplasma*, Vol. XIII, pp. 331-347, 7 plates, 1931. [Bibliography of 15 titles.]

LEWIN, LOUIS. Phantastica. Narcotic and Stimulating Drugs. *New York* (E. P. Dutton and Co.), 1931. Pp. xi + 335. 5½ × 8½ inches. $3.75.

REIMANN, STANLEY P. The hyperplastic reaction of the skin to sulphydryl and its significance in neoplasia. *American Journal of Cancer*, Vol. XV, pp. 2149-2168, 1931. [Bibliography of 20 titles.]

VI. PATHOLOGY, CLINICAL MEDICINE AND SURGERY

ANDREWS, JUSTIN. "Zebe-à-Pique" as a malariacide in Grenada, B. W. I. *American Journal of Hygiene*, Vol. XI, pp. 411-412, 1930.

ANDREWS, JUSTIN, and MOSES PAULSON. The effect of barium sulphate upon the incidence of human intestinal protozoa. *Journal of Laboratory and Clinical Medicine*, Vol. XVI, pp. 39-42, 1930. [Bibliography of 3 titles.]

ANDREWS, JUSTIN, and MOSES PAULSON. The incidence of human intestinal protozoa. With especial reference to *Endamoeba histolytica*, in the residents of the temperate zone. *American Journal of the Medical Sciences*, Vol. CLXXXI, pp. 102-106, 1931. [Bibliography of 14 titles.]

COWDRY, E. V., and ARTHUR W. HAM. The life cycle of the parasite of East Coast fever in ticks transmitting the disease. (Preliminary note.) *Science*, Vol. LXXII, pp. 461-462, 1930.

COWDRY, E. V., and S. F. KITCHEN. Intranuclear inclusions in yellow fever. *American Journal of Hygiene*, Vol. XI, pp. 227-299, 1930. [Bibliography of 55 titles.]

DYER, R. E., L. F. BADGER, and A. RUMREICH. Rocky Mountain spotted fever (eastern type). Transmission by the American dog tick (*Dermacentor variabilis*). Reprint No. 1483. *Public Health Reports*, Vol. 46, pp. 1403-1413, 1931. 5 cents.

FOLIA BIOLOGICA. Publication del personal tecnico del Instituto Bacteriologico del Departamento Nacional de Higiene. *Buenos Aires*. Nos. 1, 2, 3 and 4. April, pp. 1-4; May, pp. 5-9; June and July, pp. 10-20, 1931.

GARDNER, A. D. Microbes and Ultramicrobes. An Account of Bacteria, Viruses and the Bacteriophage. The Dial Press Monographs on Biological Subjects. (With an Appendix by G. R. deBeer.) *New York* (The Dial Press), 1931. Pp. viii + 120. 4 × 6½ inches. $1.50.

KHALIL, MOHAMED BEY. The Bibliography of Schistosomiasis (Bilharziasis), Zoological, Clinical and Prophylactic. The Egyptian University, The Faculty of Medicine, Publication No. 1. *Cairo* (Egyptian University), 1931. Pp. x+ 506. 8¼ × 9½ inches. (Paper.)

LEVINE, MICHAEL. Studies in the cytology of cancer. *American Journal of Cancer*, Vol. XV, pp. 144-211; 788-834; 1410-1494; 1931. [Bibliography of 309 titles.]

MAJOR, RALPH H. The Doctor Explains. *New York* (Alfred A. Knopf, Inc.), 1931. Pp. xxiv + 277. 5¾ × 8⅝ inches. $3.50.

OWRE, ALFRED. Is dentistry a specialty of medicine? *Dental Items of Interest*, May, 1931, 4 pp.

OWRE, ALFRED. The relationship of the physician and the dentist. *Dental Survey*, February and March 1931. Pp. (of reprint) 1-13.

PAULSON, MOSES, and JUSTIN ANDREWS. The incidence of human intestinal protozoa in duodenal aspirates. *Journal of the American Medical Association*, Vol. 94, pp. 2063-2065, 1930.

PHILIBERT, ANDRÉ. Précis de Bacteriologié Médicale. *Paris* (Masson et Cie), 1931. Pp. xi + 551. 5¼ × 7½ inches. 50 francs (paper); 65 francs (cloth).

REIMANN, STANLEY P., and FREDERICK H. SAFFORD. The avoidable delay in the treatment of carcinoma. *American Journal of Cancer*, Vol. XV, pp. 1338-1351, 1931. [Bibliography of 3 titles.]

RØNNE, HENNING. Biologische Gesetzlichkeit. *Acta Pathol. et Microbiol. Scandinavica*, Suppl. 3, pp. 343-367, 1930.

SMITHBURN, K. C., J. M. MASTERS, and L. G. ZERFAS. The treatment of secondary anemia (especially with various preparations of iron). *Journal of Laboratory and Clinical Medicine*, Vol. XVI, pp. 858-867, 1931. [Bibliography of 18 titles.]

SMITHBURN, KENNETH C., and L. G. ZERFAS. The inhibitory action of infection and fever on the hematopoietic response in the case of pernicious anemia. *Annals of Internal Medicine*, Vol. 4, pp. 1108-1111, 1931.

SMITHBURN, K. C., and L. G. ZERFAS. The neural symptoms and signs in pernicious anemia. The effects of liver extract. *Archives of Neurology and Psychiatry*, Vol. 25, pp. 1100-1110, 1931.

SVENSGAARD, ELISABETH. Blood Sugar in Normal and Sick Children with Special Reference to Coeliac Disease. *Copenhagen* (Levin und Munksgaard), 1931. Pp. 245. 6½ × 10 inches.

TSUCHIYA, H., and JUSTIN ANDREWS. A report on a case of giardiasis. *American Journal of Hygiene*, Vol. XII, pp. 297-298, 1930. [Bibliography of 3 titles.]

W. H. H. Dentistry as a specialty of medicine. *Canadian Medical Association Journal*, Vol. XXIV, pp. 845-847, 1931.

VII. METHODOLOGY

1. *Mensurational and Observational*

UNITED STATES DEPARTMENT OF JUSTICE, Bureau of Investigation. How to take fingerprints. *Washington* (U. S. Government Printing Office), 1931. Pp. iii + 8. 5⅞ × 9 inches.

2. *Mathematical and Statistical*

COATS, R. H. Enumeration and sampling in the field of the census. *Journal of the American Statistical Association*, Vol. XXVI, pp. 270-284, 1931.

GINI, C. Intorno alle curve di concentrazione. *XX^e Session de l'Institut International de Statistique, Madrid,* 1931. II, Comm., pp. (of reprint) 1-63.

ISSERLIS, L. On the moment distributions of moments in the case of samples drawn from a limited universe. *Proceedings of the Royal Society,* A, Vol. 132, pp. 586-604, 1931.

PIETRA, G. Contributions à la méthodologie du calcul du rapport de concentration. *XX^e Session de l'Institut International de Statistique, Madrid,* 1931. II, Comm., pp. (of reprint) 1-9.

RIETZ, H. L. Comments on applications of recently developed theory of small samples. *Journal of the American Statistical Association,* Vol. XXVI, pp. 150-158, 1931.

RIETZ, H. L. Some remarks on mathematical statistics. *Science,* Vol. 74, pp. 82-86, 1931.

ROESLE, E. Die Klassifikation der Geisteskrankheiten für statistische Zwecke. *Monatsschrift für Psychiatrie und Neurologie,* Bd. LXXIII, pp. 200-216, 1929.

UGGÈ, ALBINO. Osservazioni sul metodo per la misura statistica della mobilità del lavoro. *Rivista Internazionale di Scienze Sociali e Discipline Ausiliarie,* Anno 39, Vol. II, Fasc. III-IV, pp. 417-431, 1931.

3. *Other and General*

HICKS, J. ALLAN. Directory of Research in Child Development. Second Edition. Compiled for National Research Council Committee on Child Development. *Reprint and Circular Series of the National Research Council* No. 102. *Washington* (National Research Council), 1931. Pp. 74. 6⅝ × 9⅝ inches. 50 cents (paper).

JAEGER, EDMUND C. A Dictionary of Greek and Latin Combining Forms Used in Zoological Names. *Springfield, Ill.* (Charles C. Thomas), 1931. Pp. iv + 157. 4 × 7 inches. $1.50 (1 to 5 copies); $1.00 if remittance accompanies order; $1.00 in quantity lots for classes.

VIII. GENERAL AND MISCELLANEOUS

CHRIST, G. ELGIE and A. L. HAYDON, based on the original work by James Wood. The Nuttall Encyclopaedia. Dealing with Biography, Geography, History, Mythology, Literature, Science and Invention, Religions of the World, Politics, Art, Music, the Stage, Sport, Business, Law, Etc. Also Appendices Giving Populations, Pseudonyms of Writers and Artists, Characters in Drama and Fiction, etc. *New York* (Frederick Warne and Co., Inc.), 1931. Pp. vii + 695. 5½ × 8¼ inches. $3.00 net.

GUYER, MICHAEL F. Animal Biology. *New York* (Harper and Brothers), 1931. Pp. xii + 744. 5½ × 8⅜ inches. $3.75. [Bibliography of 92 titles.]

HARDING, T. SWANN. The Degradation of Science. *New York* (Farrar and Rinehart, Inc.), 1931. Pp. xi + 386. 5¾ × 8½ inches. $3.00.

PYCRAFT, W. P. (Edited by). The Standard Natural History from Amoeba to Man. Contributors: G. J. Arrow, M. Burton, W. T. Calman, J. G. Dollman, F. W. Edwards, C. C. A. Monro, J. R. Norman, H. W. Parker, W. P. Pycraft, N. D. Riley, G. C. Robson, Theodore H. Savory. *New York* (Frederick Warne and Co., Inc.), 1931. Pp. xiv + 942. 6¼ × 8¾ inches. $6.00.

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